TX2100: A Differentiated Anti-Angiogenic Therapy for HHT and Other Bleeding Disorders

FEBRUARY 24, 2026

TECTONIC Therapeutic

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These forward-looking statements reflect our current beliefs and expectations. Many factors may cause differences between current expectations and actual results, including the early stage of our development efforts; success in preclinical testing and earlier clinical trials does not ensure that later clinical trials will generate the same results or otherwise provide adequate data to demonstrate the efficacy and safety of a product candidates; clinical site activation rates or clinical trial enrollment rates that are lower than expected; changes in expected or existing competition; changes in the regulatory environment; the uncertainties and timing of the regulatory approval process; the impact of macroeconomic conditions, including the conflict in Ukraine and the conflict in the Middle East, heightened inflation and uncertain credit and financial markets, on our business, clinical trials and financial position; and unexpected litigation or other disputes. These and other risks are described more fully in our filings with the Securities and Exchange Commission (“SEC”), including the risks detailed in our Quarterly Report on Form 10-Q filed with the SEC on November 6, 2025, and other documents we subsequently filed with or furnished to the SEC. All forward-looking statements contained in this presentation speak only as of the date on which they were made. Except as required by law, we assume no obligation to update any forward-looking statements contained herein to reflect any change in expectations, even as new information becomes available.

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Agenda

Welcome and Introduction	**Alise Reicin, MD** Chief Executive Officer, Director
HHT: The Disease and Unmet Need	**Hanny Al-Samkari, MD** Mass. General Hospital, Harvard Medical School
TX2100: Discovery and Rationale	**Peter McNamara, PhD** Chief Scientific Officer
TX2100: Clinical Update	**Marcie Ruddy, MD** Chief Medical Officer
Questions and Answers	

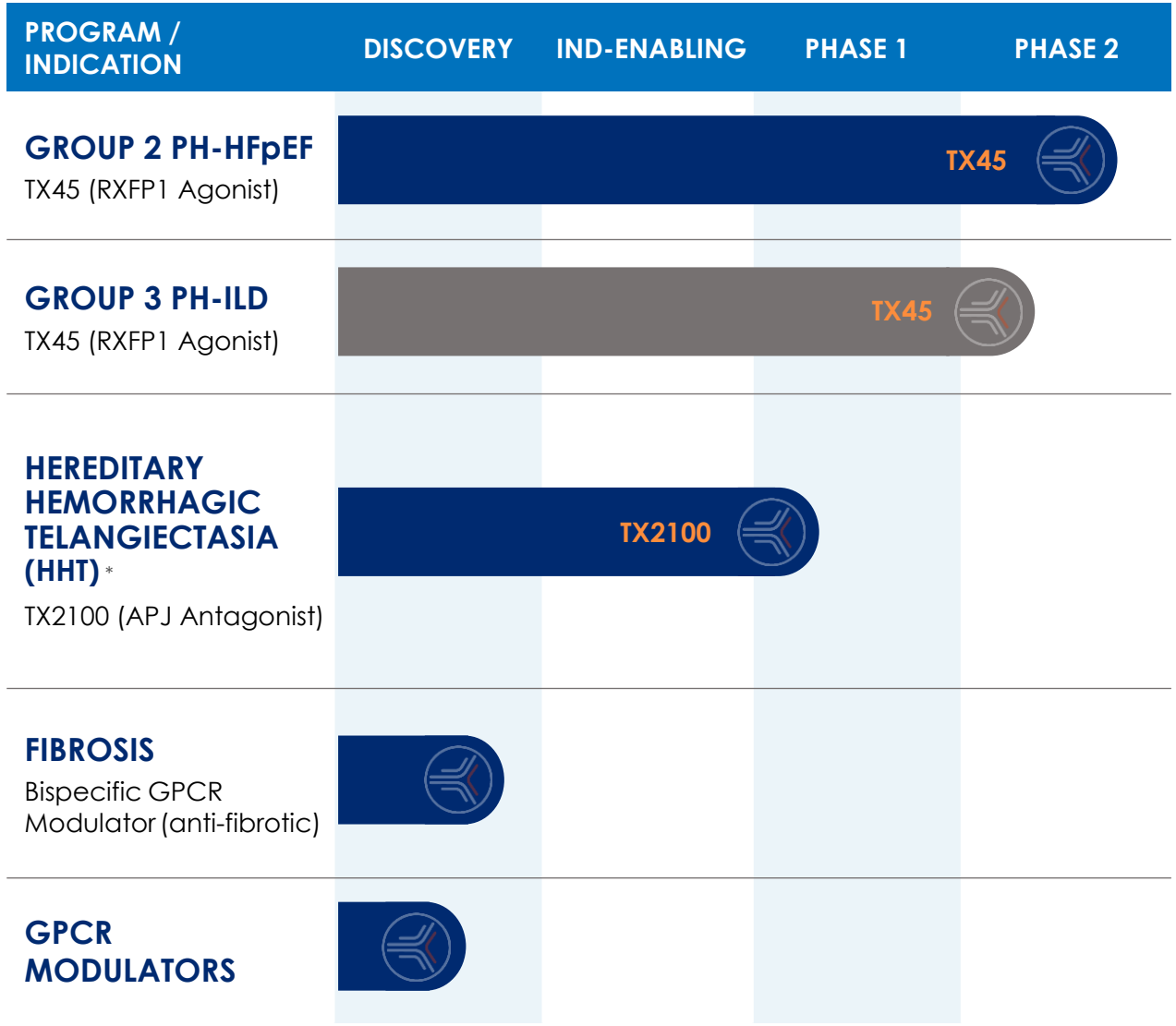
Advancing a High-Value Pipeline of GPCR-Targeted Therapies

Clinical-stage biotech
- Biologics to target GPCRs

Significant therapeutic opportunities
- Diseases with high unmet need and limited options

Robust, multi-product pipeline
- Two clinical programs, three indications, and an emerging preclinical pipeline
- TX45 being explored in Phase 2 for both Group 2 PH HFpEF and PH-ILD
- TX2100 for Hereditary Hemorrhagic Telangiectasia and other bleeding disorders

Company with clinical momentum
- Well-capitalized to advance high-value pipeline



** Subject to positive Phase 1 data*

TECTONIC Therapeutic

TX2100 for Hereditary Hemorrhagic Telangiectasia (HHT)

Blockbuster Potential	• HHT is a genetic disorder of dysregulated angiogenesis leading to recurrent bleeding, anemia, arteriovenous malformations (AVMs) and reduced life expectancy with no approved therapies
Orphan Indication	• Estimated ~75K HHT patients in the US; anti-angiogenic drugs (e.g., bevacizumab, pomalidomide) reduce bleeding but chronic use limited by toxicity
APJ: The GPCR Target for the Hormone Apelin	• Highly selective/specific anti-angiogenic target. APJ expressed mainly in endothelial cells, Apelin/APJ pathway is usually quiescent and upregulated during pathologic angiogenesis for greater selectivity vs. other anti-angiogenic agents – Potential to expand into a broader group of bleeding disorders caused by dysregulated angiogenesis
TX2100	• A potential first-in-class APJ antagonist with subcutaneous administration designed to treat HHT with anticipated benefit of anti-angiogenic therapy with improved safety
Preclinical to Clinical Translation	• Anti-angiogenic agents demonstrate activity both in HHT preclinical models and in patients • Efficacy of TX2100 shown in two HHT preclinical models, increasing probability of success
TX2100 Phase 1 Study Initiated	• Phase 1a healthy volunteer clinical trial ongoing

Today's Call Features Dr. Hanny Al-Samkari, Joined by Company Management

Hanny Al-Samkari, MD

Dr. Al-Samkari is the Peggy S. Blitz Endowed Chair in Hematology/Oncology at the Massachusetts General Hospital and an Associate Professor of Medicine at Harvard Medical School. He is a classical hematologist and NIH-funded clinical investigator and serves as the Co-Director of the MGH HHT Center of Excellence. He is the current Chair of the Cure HHT Global Research and Medical Advisory Board. His clinical and research interests are in hemostasis, thrombosis and hemolysis, with focuses in HHT and other bleeding disorders. He is an internationally recognized expert in the clinical development of novel therapeutics for these disorders and serves as the principal investigator for many clinical trials. Dr. Al-Samkari cares for several hundred patients with HHT and has clinics dedicated to the care of patients with HHT.

Alise Reicin, M.D.
CEO, Director

Peter McNamara, Ph.D.
CSO

Marcella Ruddy, M.D.
CMO

A Teaching Affiliate
of Harvard Medical School

Unmet Need in Hereditary Hemorrhagic Telangiectasia

Hanny Al-Samkari, M.D.
The Peggy S. Blitz Endowed Chair in Hematology/Oncology
Classical Hematologist and Clinical Investigator
Co-Director, HHT Center of Excellence
Massachusetts General Hospital
Associate Professor of Medicine
Harvard Medical School

MASSACHUSETTS
GENERAL HOSPITAL

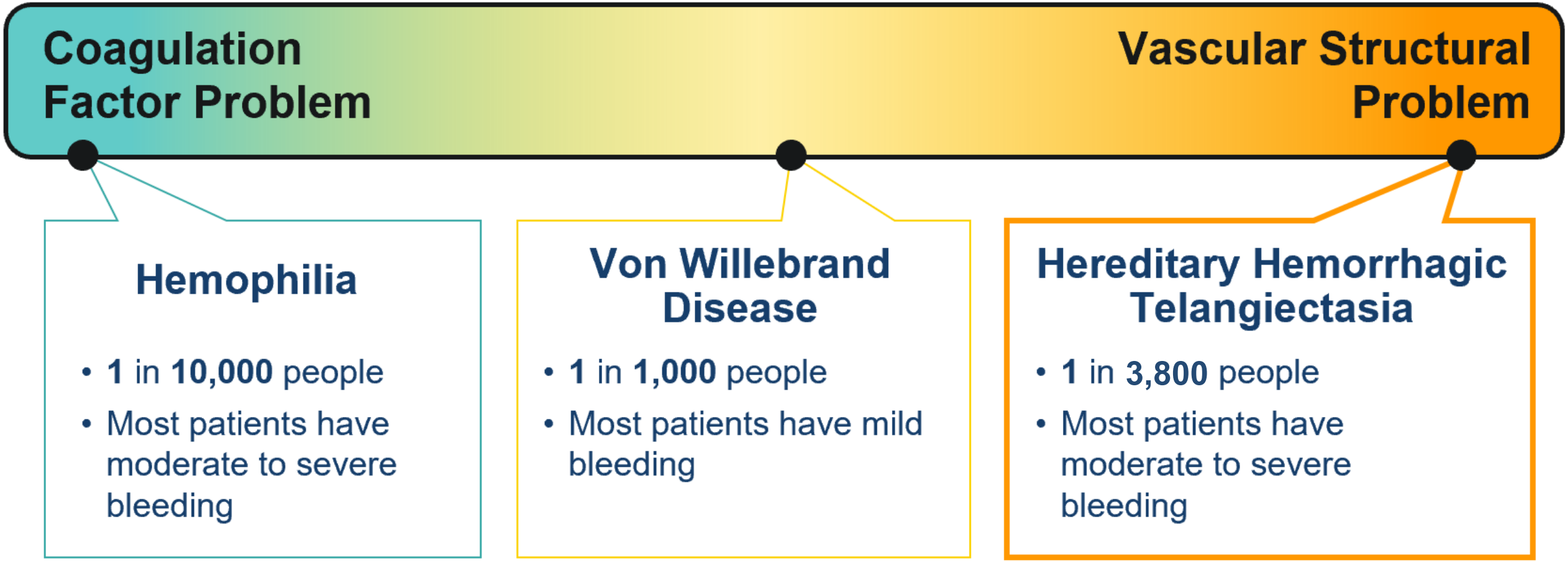
Typical Patient Case

- 41-year-old man with severe nosebleeds and chronic intestinal bleeding working in the biomedical field
- Diagnosed with HHT in his 20s, but not cared for at HHT center; sent for regular nasal and intestinal cautery procedures that each worked for a couple of months but provoked worse nosebleeding as time went on
- Ultimately went on disability and career halted because of:
  - Constant blood gushing from his face limiting him at work
  - Chronic severe anemia despite regular intravenous iron and blood transfusions
  - Constant ER visits for severe nosebleeds and hospitalizations from severe intestinal bleeding
  - Diagnosis of major depressive disorder from nosebleeding; started on antidepressant which worsened his bleeding (prescribing doctor did not recognize this as a side-effect of the antidepressant)
- Then saw me at the MGH HHT CoE; “I am barely 40 but I feel like my life is nearly over. I just want to go back to work, and maybe one day be able to have a girlfriend.”

Another Typical Patient Case

- 37-year-old man, father of three children, diagnosed with HHT one month prior to his visit
- Came from Maine to HHT Center of Excellence at MGH in Boston
- Gushing nosebleeds and chronic intestinal bleeding causing severe anemia, resulting in severe fatigue, reducing work hours, threatened employment (works in a construction job), ability to care for family
- One son died of a brain hemorrhage at birth; another son had a brain hemorrhage shortly after birth but lived with severe disability
- Daughter has recurrent nosebleeds causing anxiety, distress, social isolation at school

The Spectrum of Inherited Bleeding Disorders



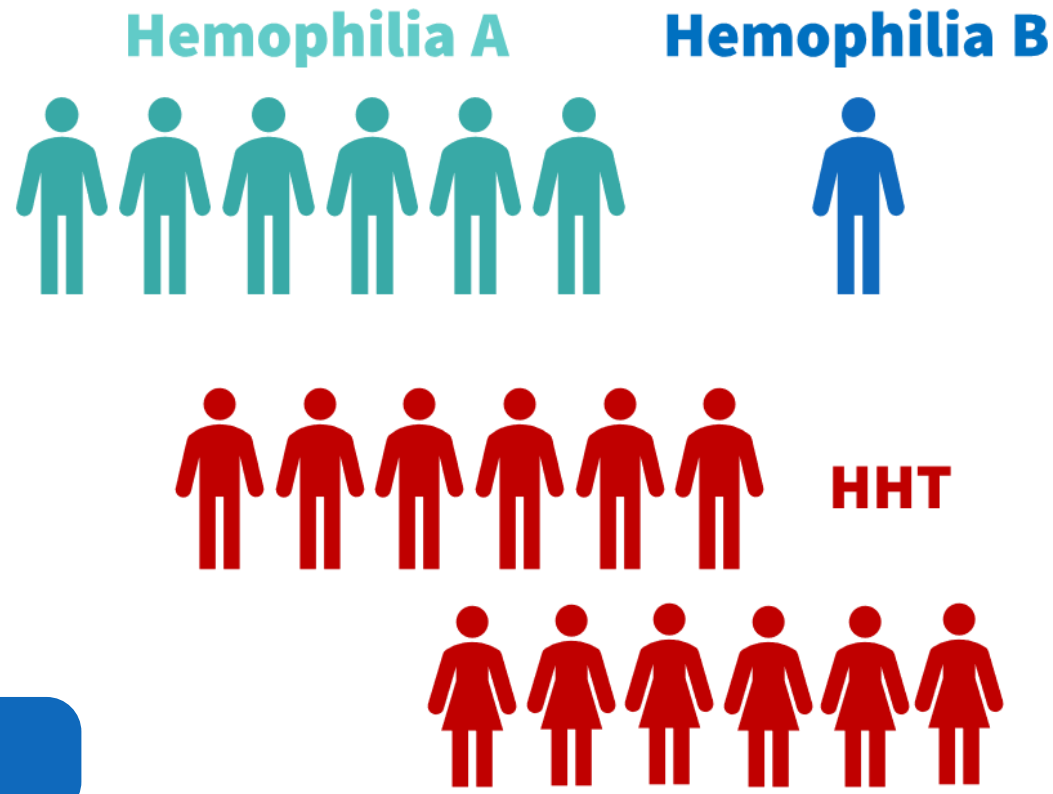
HHT is a Multisystem Hereditary Bleeding Disorder with Numerous Morbid and Potentially Fatal Manifestations

- Progressive, multisystem bleeding disorder due to abnormal vessel formation
  - Mucocutaneous telangiectasias → **severe recurrent epistaxis** and **chronic gastrointestinal hemorrhage**
  - **Iron deficiency anemia**, often **iron infusion and RBC transfusion-dependent**
  - Visceral and CNS arteriovenous malformations (AVMs) in **lung, liver, brain,** others
    - Hemorrhagic and embolic stroke
    - Liver disease and cirrhosis
    - Pulmonary hypertension, pulmonary hemorrhage
    - High output heart failure
- Patients rank ***bleeding*** as most important clinical manifestation (by a wide margin)
  - AVMs and anemia tie for second
- ***No approved therapies worldwide to date***

Al-Samkari H., *Blood* 2021; Al-Samkari H, *Blood* 2024

HHT is the Second-Most-Common Inherited Bleeding Disorder

- **Autosomal dominant** inheritance, 1 in 3800 people
- Occurs in all sexes equally
- Most clinically significant and morbid inherited bleeding disorder of women
- Patients with HHT have **reduced overall survival** compared with healthy controls
- ~80,000 people with HHT in US



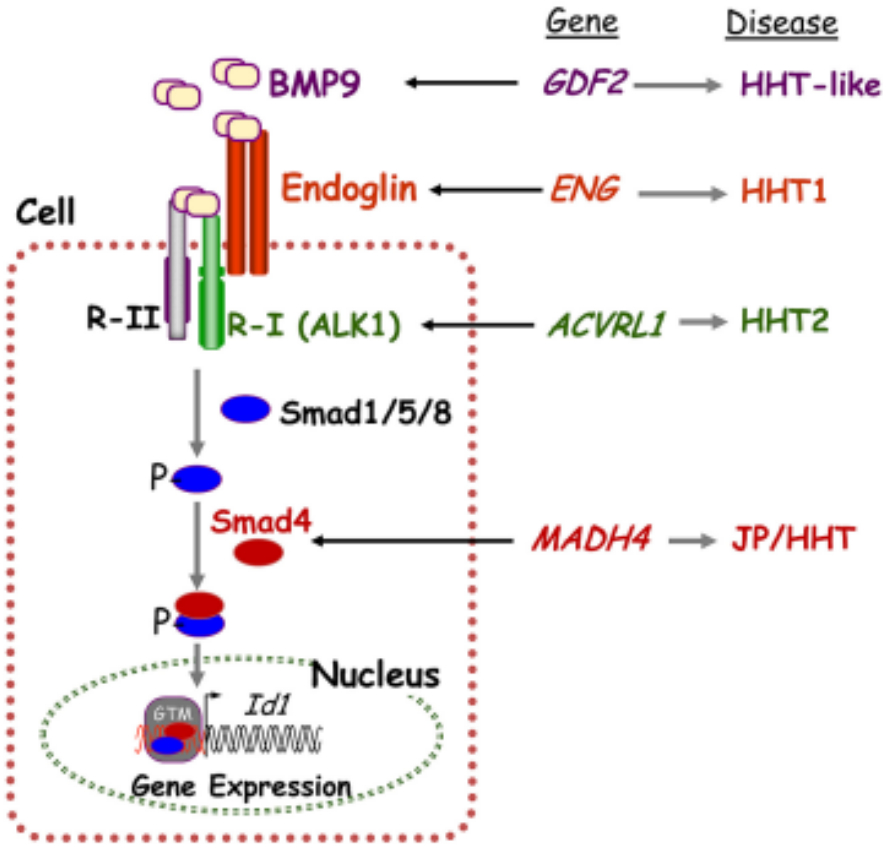
HHT Affects 1.6 Million Worldwide

Al-Samkari H., *Blood* 2021; Zhang et al., *Blood Advances* 2024

HHT is Caused by Mutations in Genes in the BMP9/ALK1 Pathway

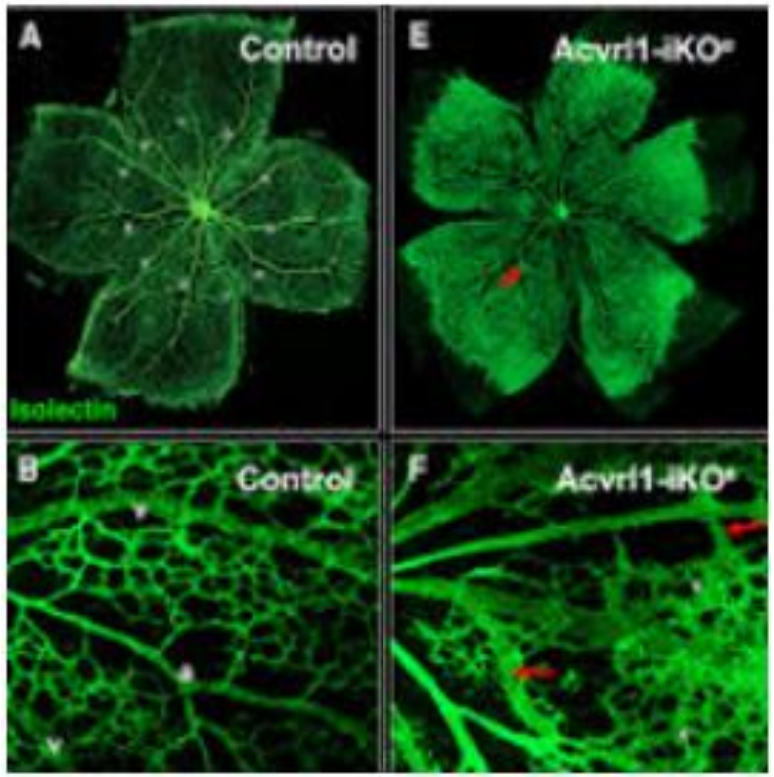
- **Genetic Drivers of HHT**
  - HHT arises from **loss-of-function mutations** in key vascular-signaling genes:
  - BMP9 (*GDF2*), **Endoglin** (*ENG*), **ALK1** (*ACVRL1*), and SMAD4 (*MADH4*)
  - These mutations disrupt BMP9/ALK1 signaling, a pathway **required** for **vascular quiescence** and **controlling angiogenesis**

- **Consequences of BMP9/ALK1 pathway loss**
  - Loss of this pathway shifts **endothelial cells** into a **persistent pro-angiogenic state**, driving abnormal vessel growth and AVM formation



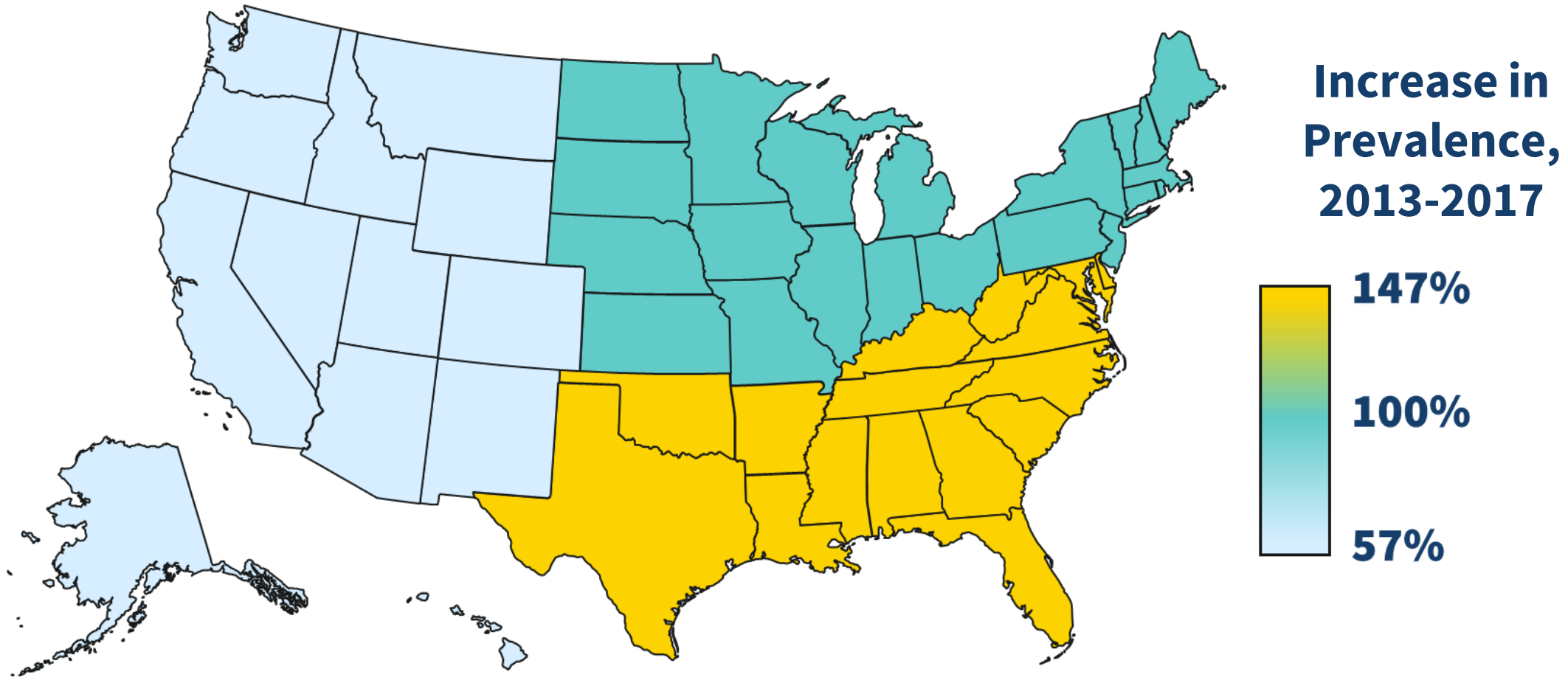
Mouse Models of HHT Replicate Disease, Are Predictive of Clinical Efficacy

- **Multiple mouse models of HHT**
  - Anti-BMP9/10 immunoblocked neonatal model
  - Endoglin (*ENG*) inducible knockout (iKO) mouse
  - SMAD4 (*MADH4*) iKO mouse
  - ALK1 (*ACVRL1*) iKO mouse (most severe model with profound GI bleeding)
- **Response to drug in mouse model predicts clinical efficacy**
  - Mouse models of HHT have a phenotype similar to human disease, with GI bleeding and AVMs in numerous locations
  - Drug efficacy in mouse models predicts human response (bevacizumab[1], pazopanib[2], and thalidomide[3] have efficacy in mouse models and in humans with HHT)



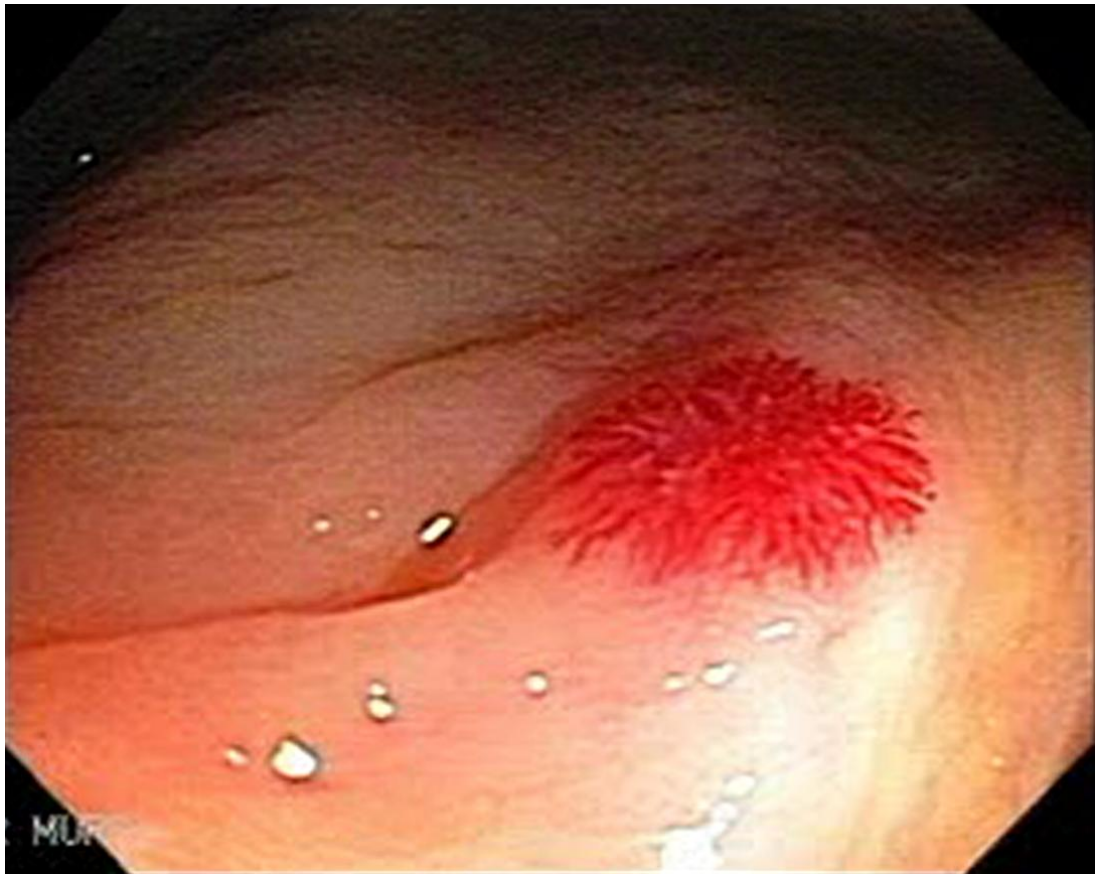
[1]Walker EJ et al. Stroke. 2012; [2]Kim YH et al. J Thromb Haemost. 2017; [3]Lebrin F et al. Nat Med 2010.

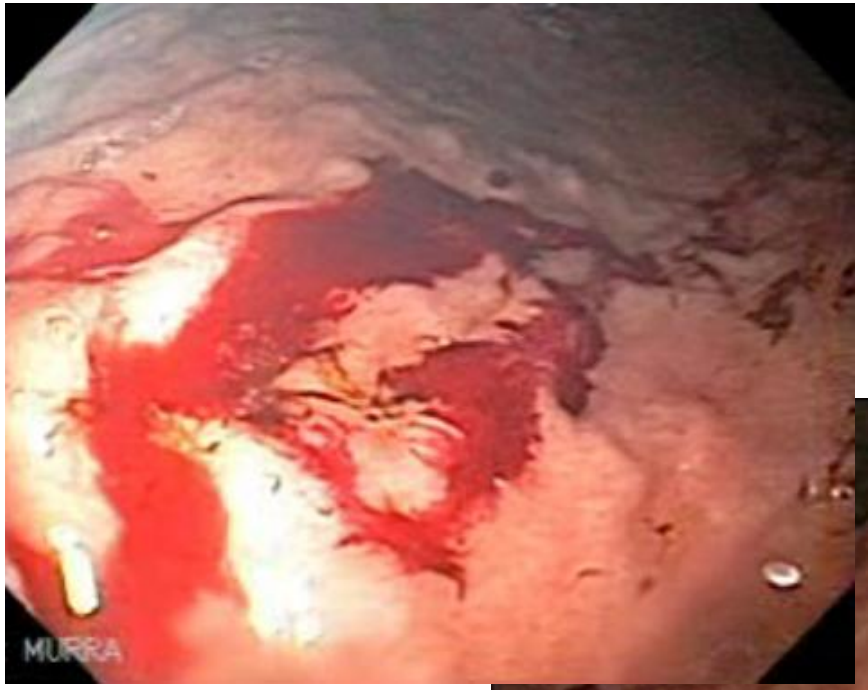
Prevalence is Increasing Because More People Are Getting Diagnosed

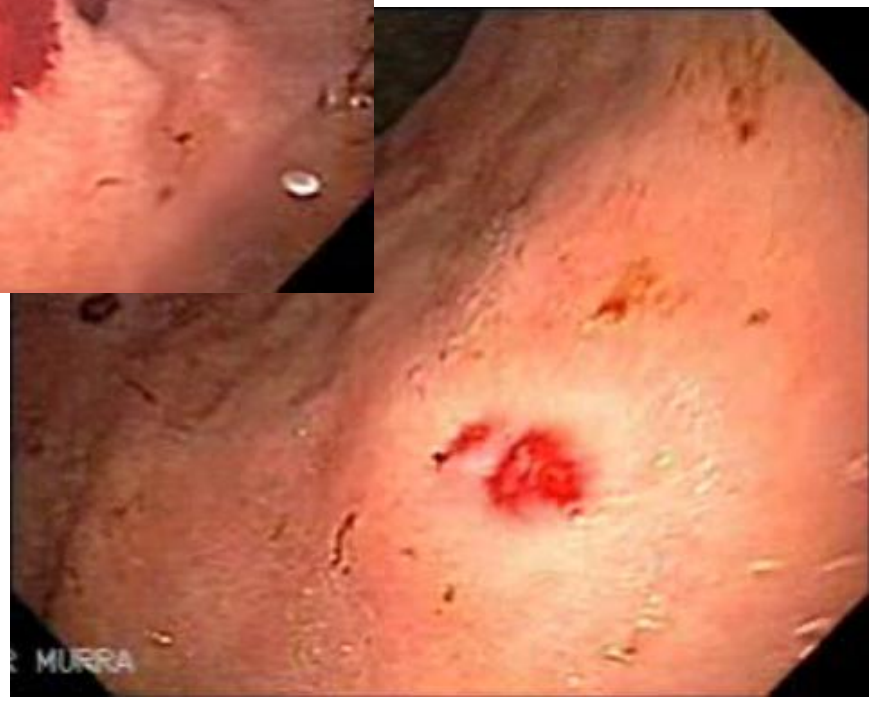


Ferry AM, et al. *Am J Rhinol Allergy*. 2020.

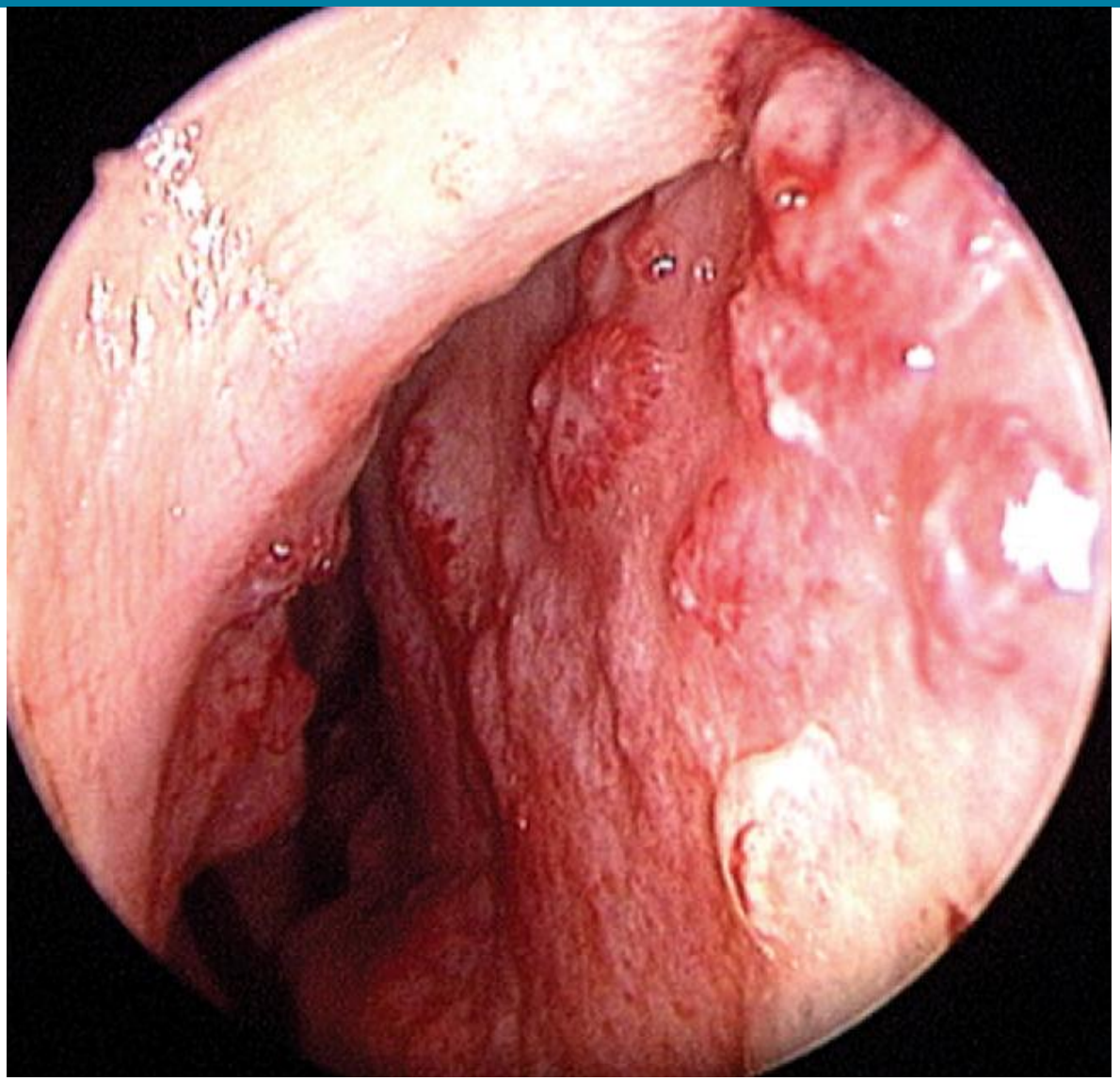
Mucocutaneous Telangiectasias: Gastrointestinal Tract

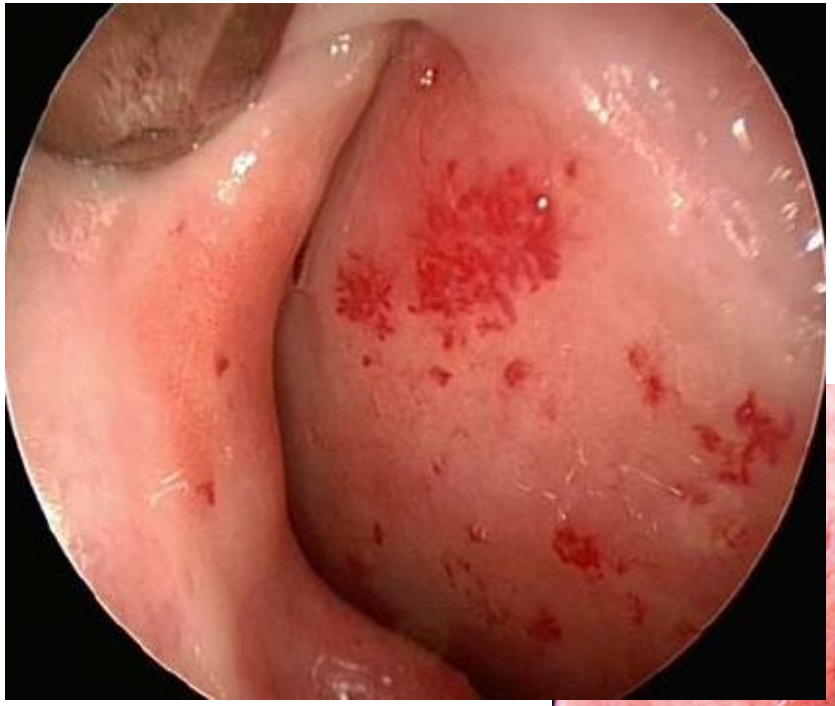


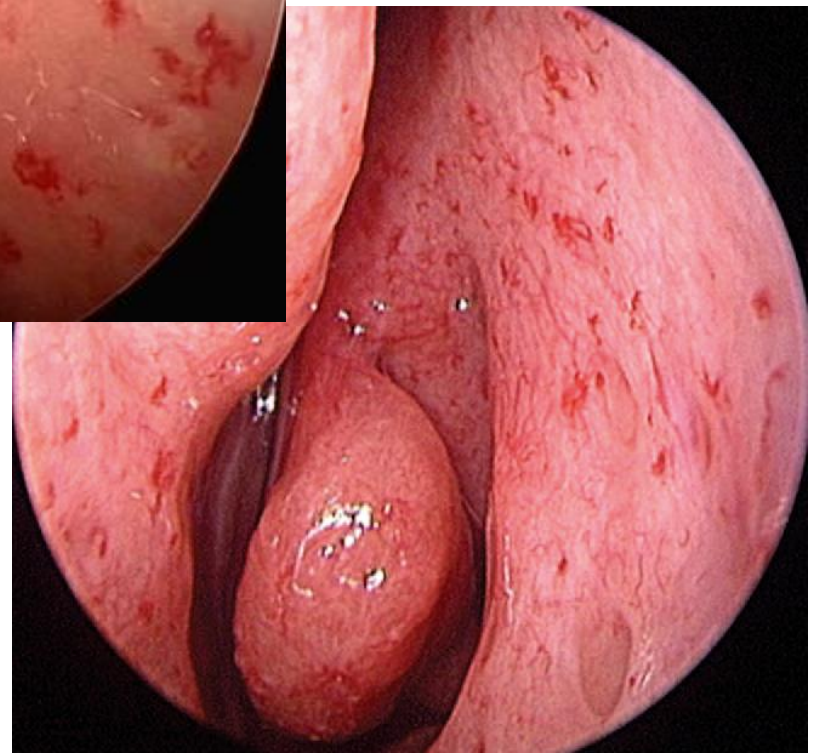




Mucocutaneous Telangiectasias: Nasal Cavity

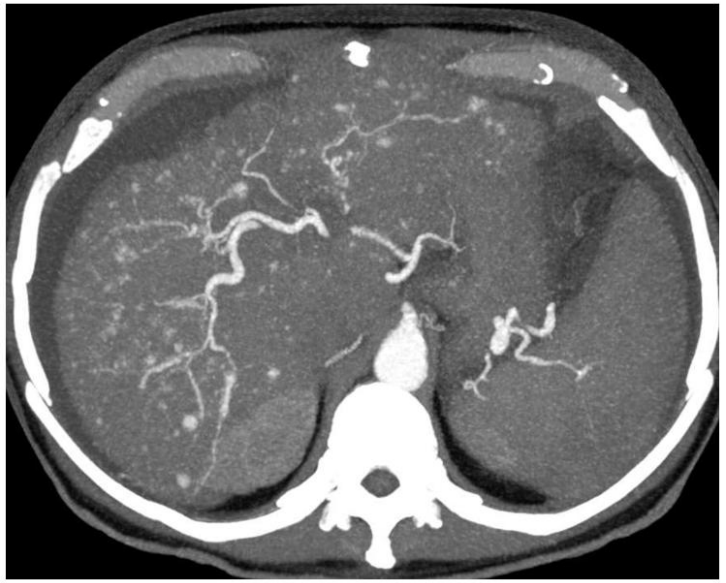




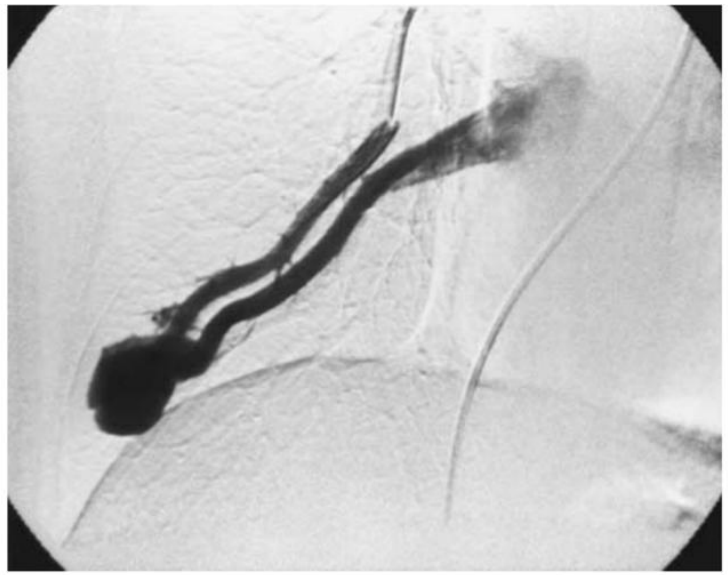


Arteriovenous Malformations (AVMs)

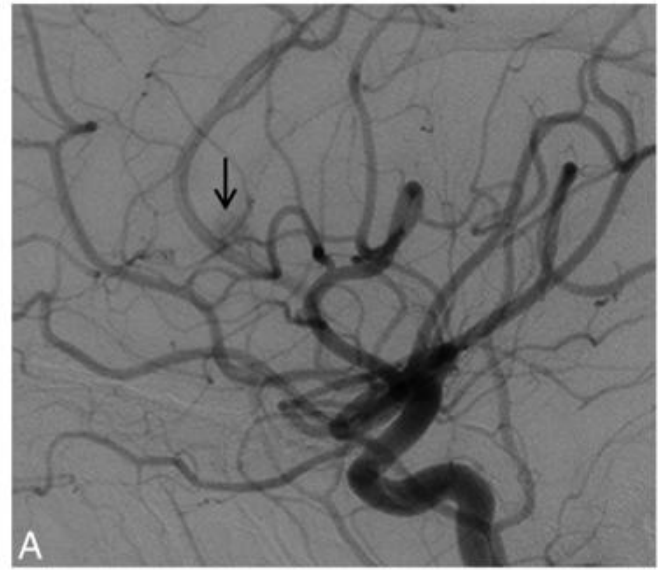
Liver



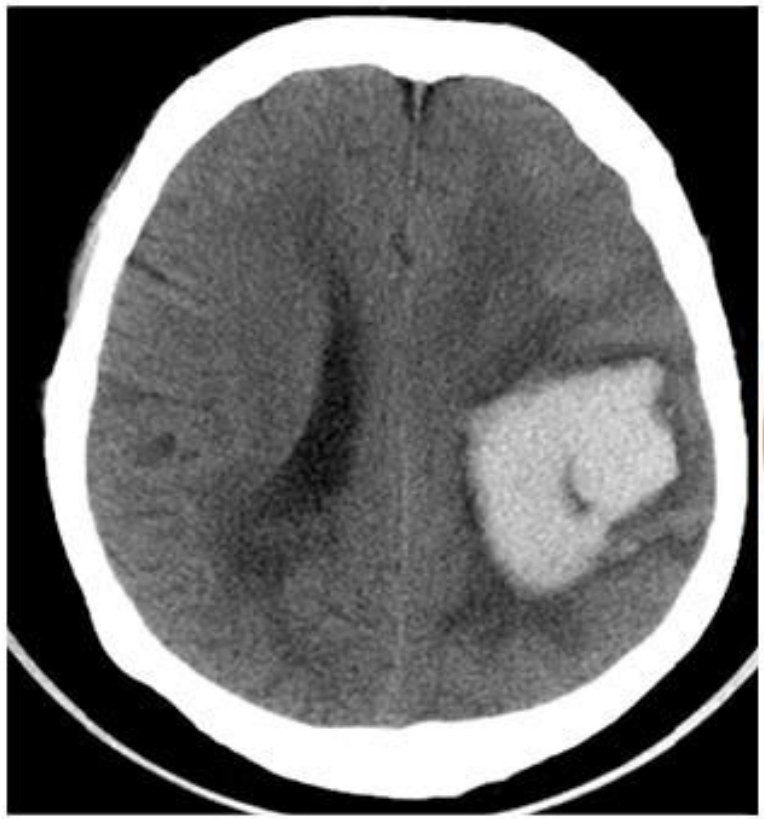
Lung

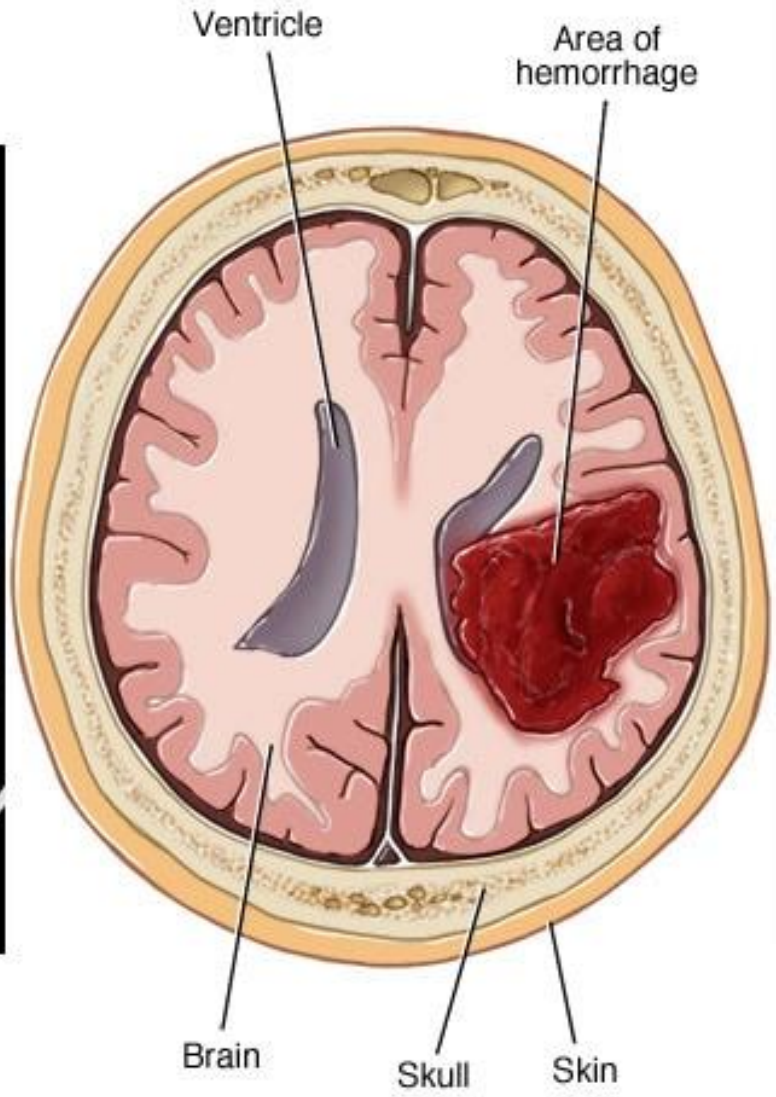


Brain



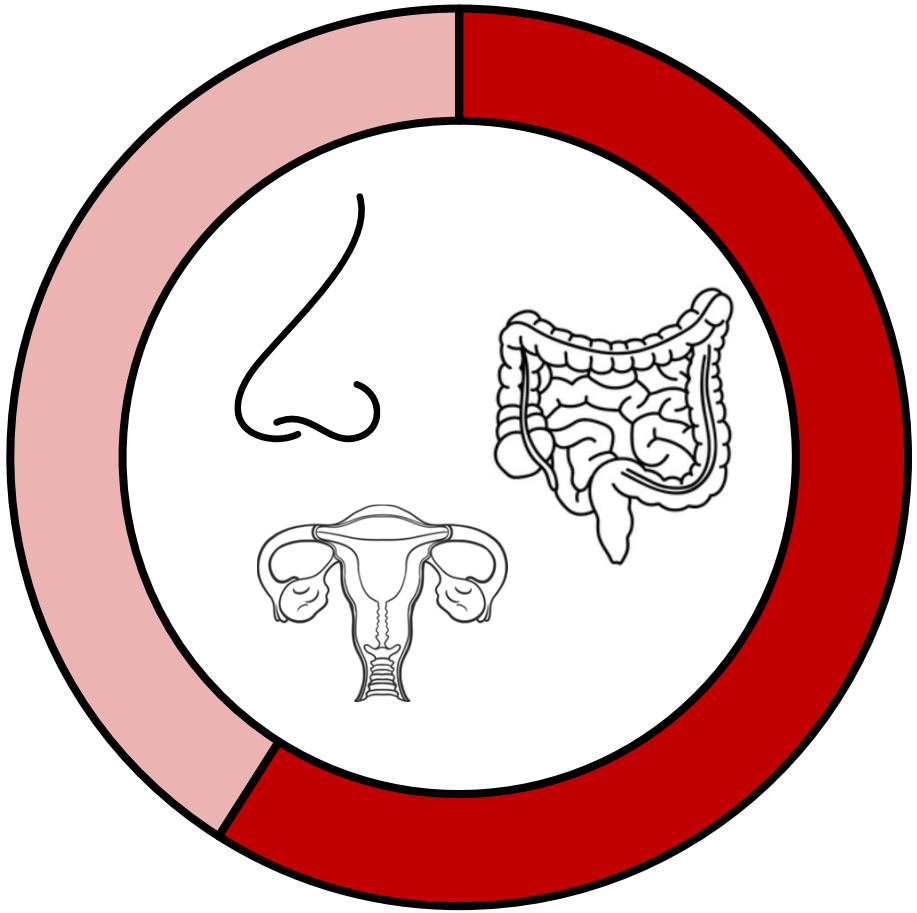
What is the MOST FEARED Complication of Any Bleeding Disorder?





Incidence of Moderate-to-Severe HHT-Associated Mucosal Bleeding in Centers of Excellence

Mild
24%

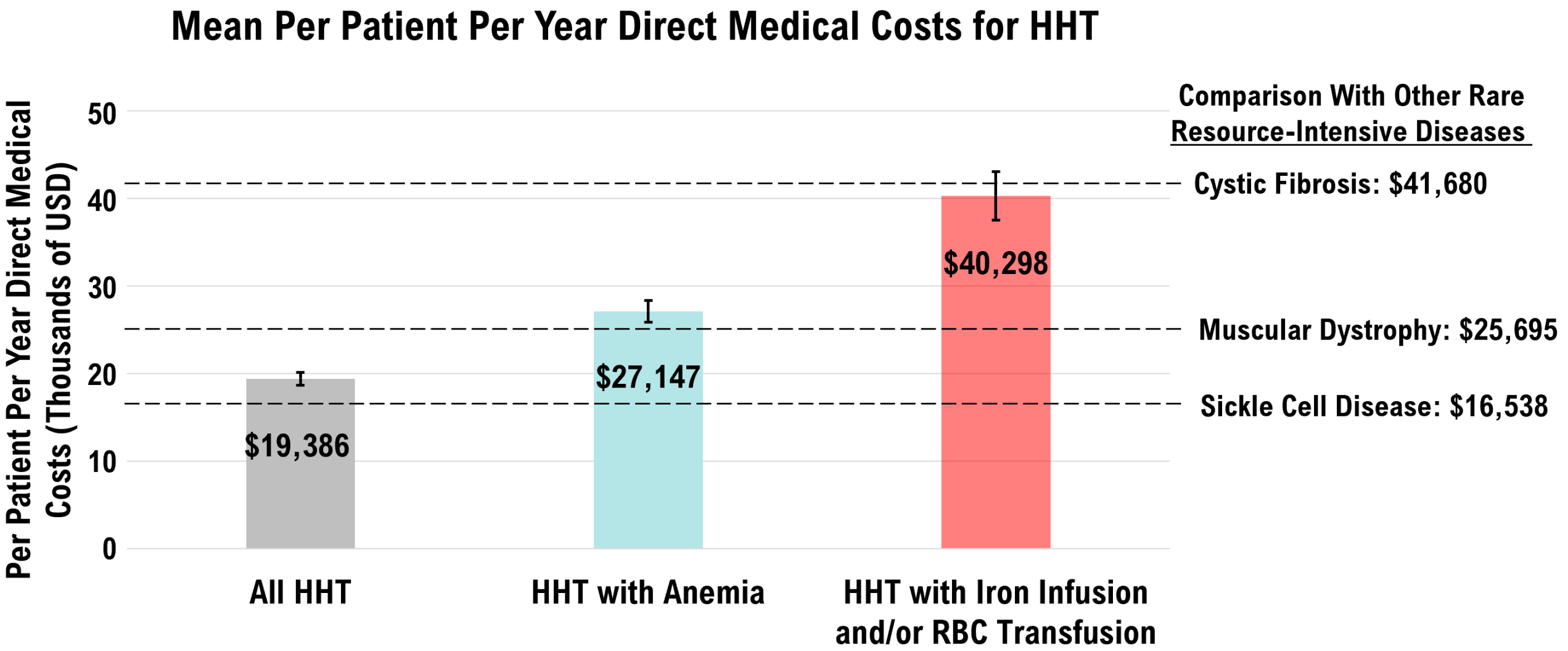


Mod-Severe
76%

Moderate-to-Severe HHT Mucosal Bleeding:
(1) ESS>4.00,
(2) Systemic medical or surgical intervention for epistaxis and/or GI bleeding,
(3) intravenous iron and/or red cell transfusion to manage anemia

Al-Samkari et al., *Blood* 2025

HHT is an Expensive Disease



Al-Samkari et al., *American Journal of Hematology* 2025

HHT is an Expensive Disease

~$500M
per year in one sample

~$2B
per year estimated total U.S. cost

12% of all diagnosed patients are **hospitalized at least once per year**

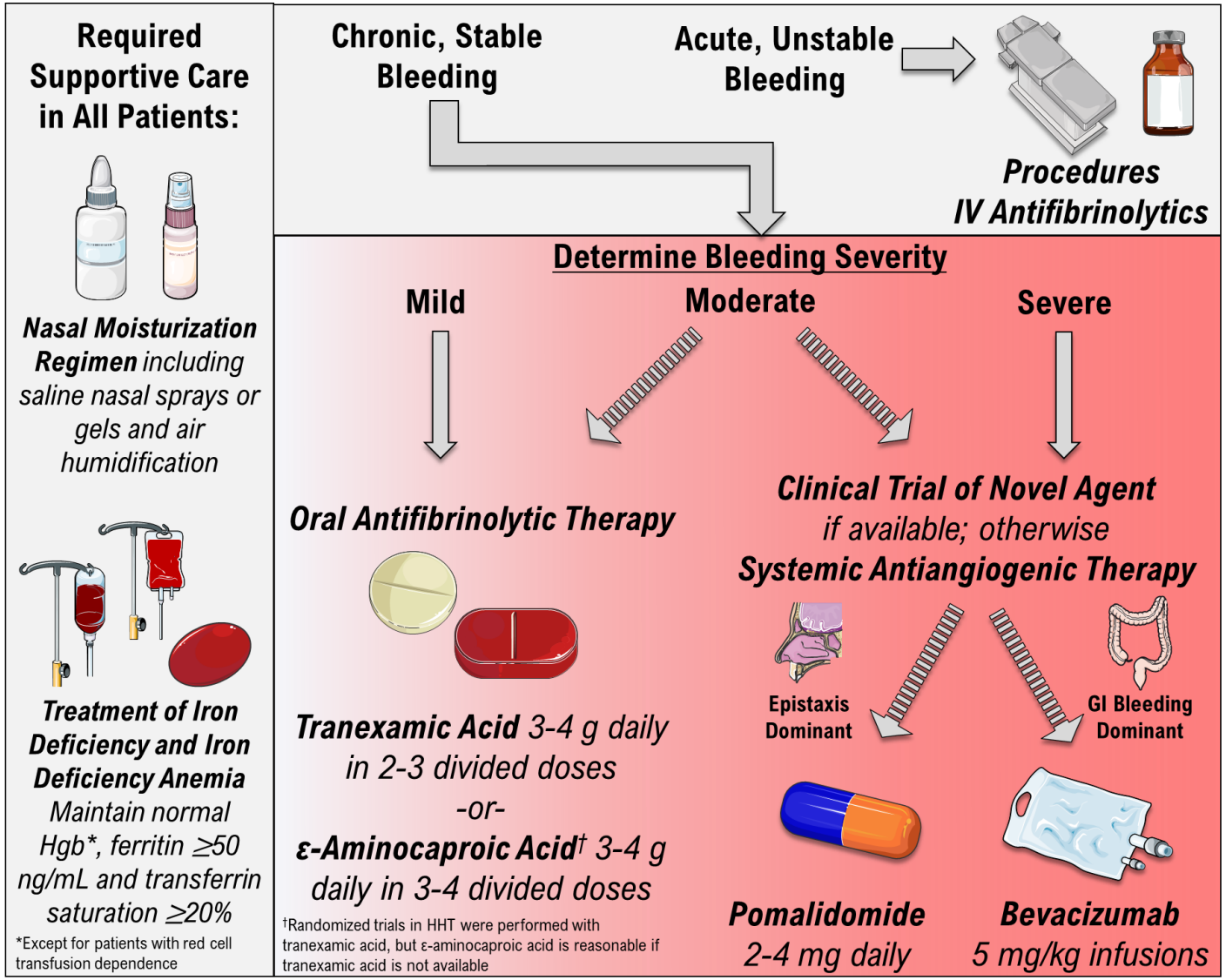
$21M spent on **one patient** treated outside of an HHT Center on huge amounts of a (wrong) expensive medication to treat bleeding in 1 year

Al-Samkari et al., *American Journal of Hematology* 2025

Many New and Striking Findings from First CHORUS Report (*Comprehensive HHT Outcomes Registry of the United States*)

- ***3 in 4*** people with HHT develop moderate-to-severe mucosal bleeding, including epistaxis, gastrointestinal, and/or heavy menstrual bleeding
- ***1 in 3*** people with HHT develop chronic GI bleeding
- ***1 in 3*** menstrual-age women with HHT develop heavy menstrual bleeding
- ***7 in 10*** people with HHT develop iron deficiency and/or anemia
- ***1 in 4*** people with HHT develop severe enough anemia to merit RBC transfusion
- ***1 in 50*** people with HHT develop pulmonary hemorrhage
- ***1 in 30*** people with HHT develop intracranial hemorrhage
- ***1 in 10*** people with HHT develop arterial thromboembolism
- ***1 in 10*** people with HHT develop serious cardiopulmonary complications (PH and/or HF)
- ***1 in 5*** people with HHT develop serious CNS complications (bAVM, stroke, ICH, epilepsy)

Current Treatment Paradigm in HHT is Deeply Inadequate



Al-Samkari H. How I Treat Bleeding in Hereditary Hemorrhagic Telangiectasia. *Blood* 2024

Limited Tolerability of Currently Used Anti-Angiogenic Drugs

Bevacizumab: Limited by hypertension, proteinuria, thromboembolism risk, waning efficacy

Pomalidomide: Limited by neutropenia, rash, neurologic side effects, constipation, thromboembolism risk, waning efficacy

No marketed drugs, including bevacizumab and pomalidomide, are currently approved by the FDA for the treatment of HHT

Al-Samkari H. How I Treat Bleeding in Hereditary Hemorrhagic Telangiectasia. *Blood* 2024

TX2100

A Differentiated Anti-Angiogenic Therapy for HHT

Peter McNamara, Ph.D.

Chief Scientific Officer

TX2100: A Potential First-in-Class APJ Antagonist for HHT and Angiogenesis-Driven Bleeding

Validated approach

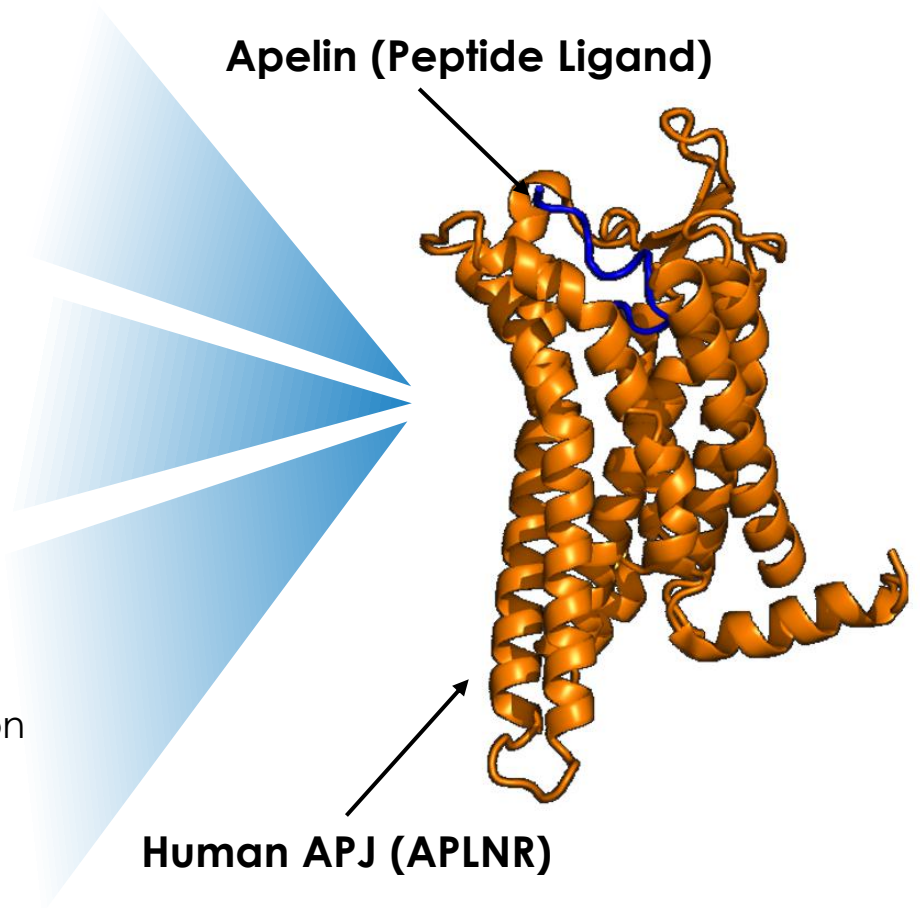
- Anti-angiogenesis reduces bleeding in HHT and related bleeding disorders, but no approved therapies
- Toxicity of oncology anti-angiogenic agents are challenging for chronic use

Differentiated target

- APJ is endothelial-enriched and apelin/APJ pathway is activated during abnormal angiogenesis
- TX2100 is designed to deliver anti-angiogenic efficacy with improved safety

De-risked translation and path to value

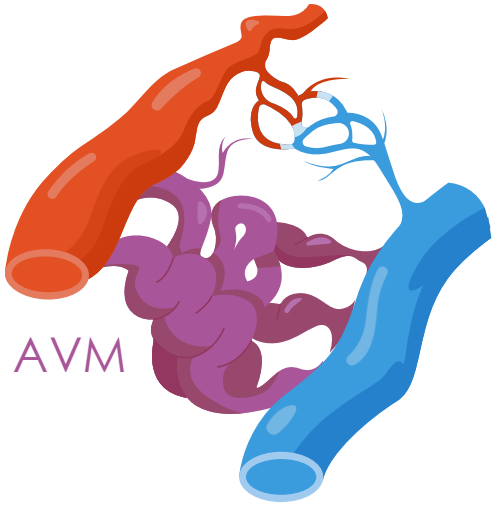
- Demonstrated preclinical activity in two validated HHT models with normalization of vasculature in model of severe disease
- Clean NHP GLP tox and durable non-clinical PK
- Phase 1a ongoing, Phase 1b and Phase 2 proof-of-concept planned



APJ is a Highly Selective, Highly Specific Anti-Angiogenic Target

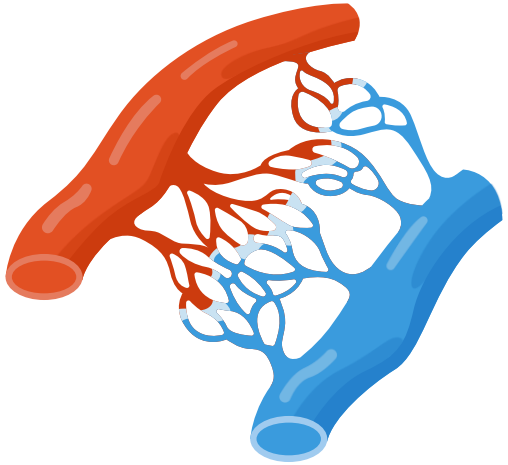
- APJ is an endothelial-enriched GPCR
- Apelin/APJ pathway is upregulated in pathological sprouting angiogenesis
- Low baseline apelin/APJ activity during normal vascular homeostasis

Upregulated apelin/APJ signaling



APJ antagonism

Blocked apelin/APJ signaling



Three Decades of Progress Lead to Convergence on APJ Antagonism to Treat HHT

Angiogenesis emerges as a key driver of HHT

1994 - 1996
ENG / ACVRL1 establish angiogenic etiology of HHT

2011
First anti-angiogenic clinical signal (bevacizumab)

2017-2018
Real-world data: anti-VEGF improves bleeding/ anemia

2019-2021+
Multiple anti-angiogenic modalities show efficacy

2025
HHT broadly accepted as dysregulated angiogenesis

APJ/apelin biology converges with HHT angiogenesis

1993 - 1998
Apelin/APJ axis identified

2004
Apelin/APJ defined as pro-angiogenic signaling axis

2004–2021: Pharma focus on APJ *agonism* (CV/HF) Limited translation beyond Phase 1

2023
Apelin is part of a conserved angiogenic signature in HHT models

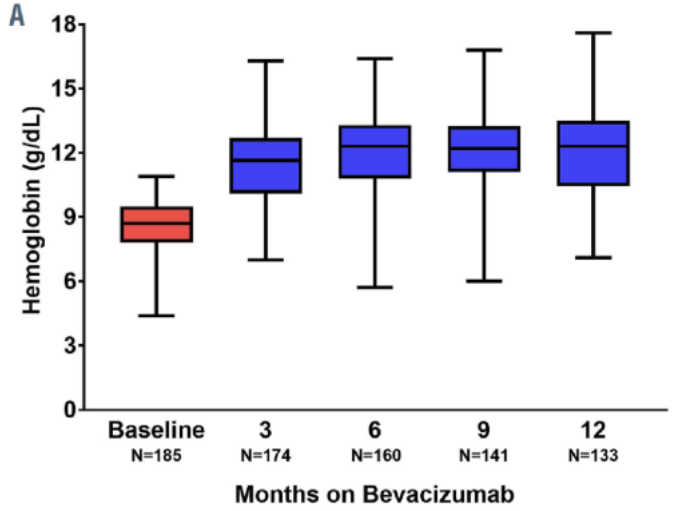
TECTONIC Therapeutic

TX2100

Tectonic recognized that HHT biology reframes APJ as a target for inhibition — leading to TX2100

Anti-Angiogenesis (VEGF blockade) is Clinically Effective in HHT but On-Target Toxicities Limit Long-Term Use

Anti-VEGF improves hemoglobin in severe HHT anemia



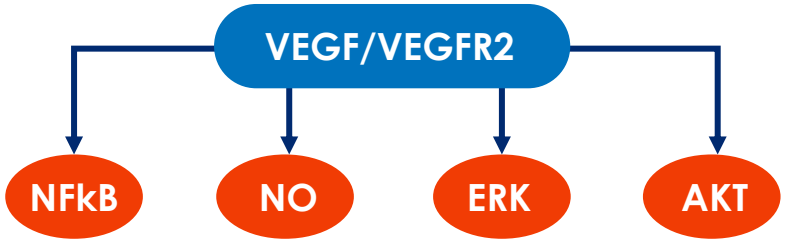
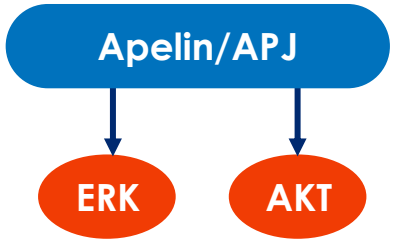
Mechanistic proof-of-concept for anti-angiogenesis comes from use of oncology drugs where anti-VEGF therapies show reduced bleeding, increased hemoglobin and less need for transfusions

Problem: Those drugs were not designed for long-term use in a non-malignant vascular disease

Solution: Develop an APJ antagonist for treatment of HHT and other angiogenesis-driven disorders that captures the benefit of anti-angiogenic therapy with improved safety

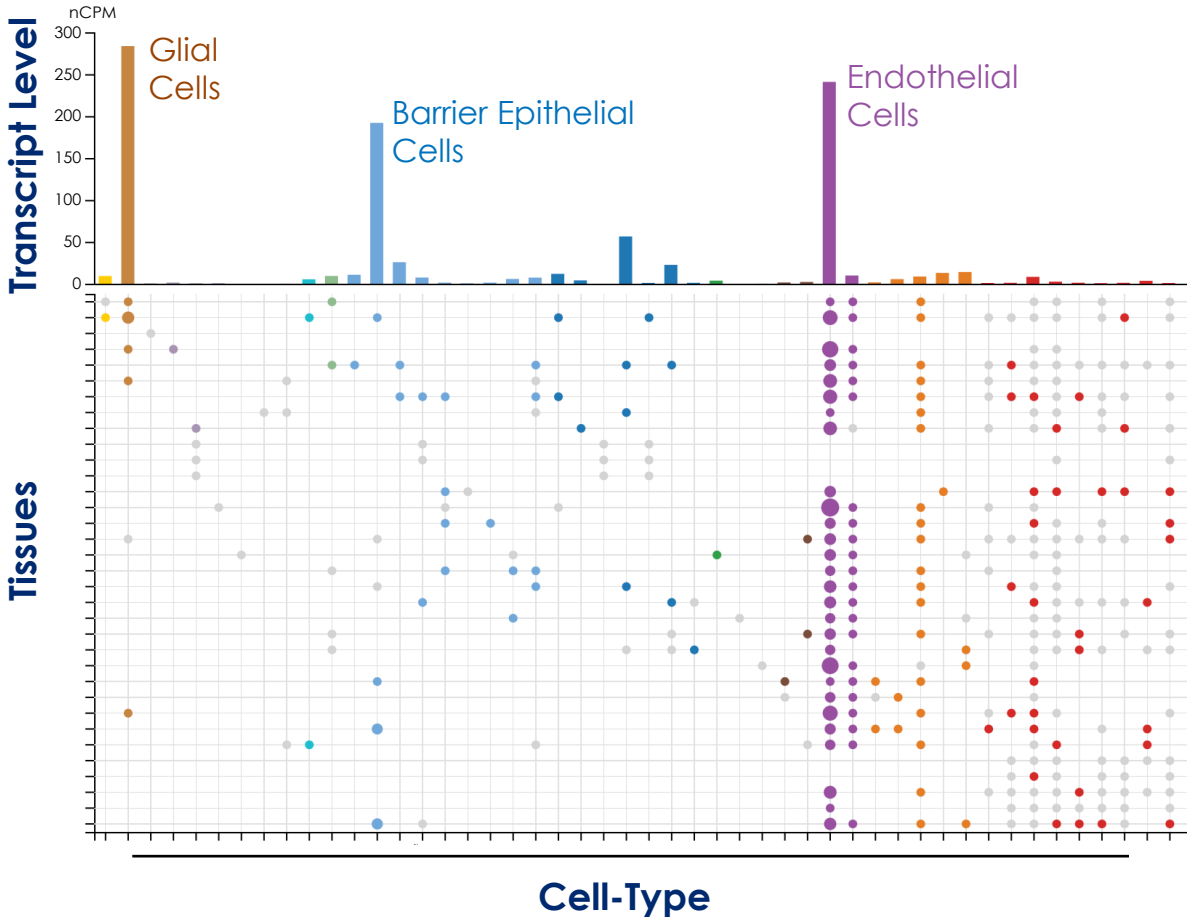
Haematologica. 2021 Aug 1; 106(8): 2161–2169

APJ Antagonist: A More Selective & Tolerable Anti-Angiogenic Agent

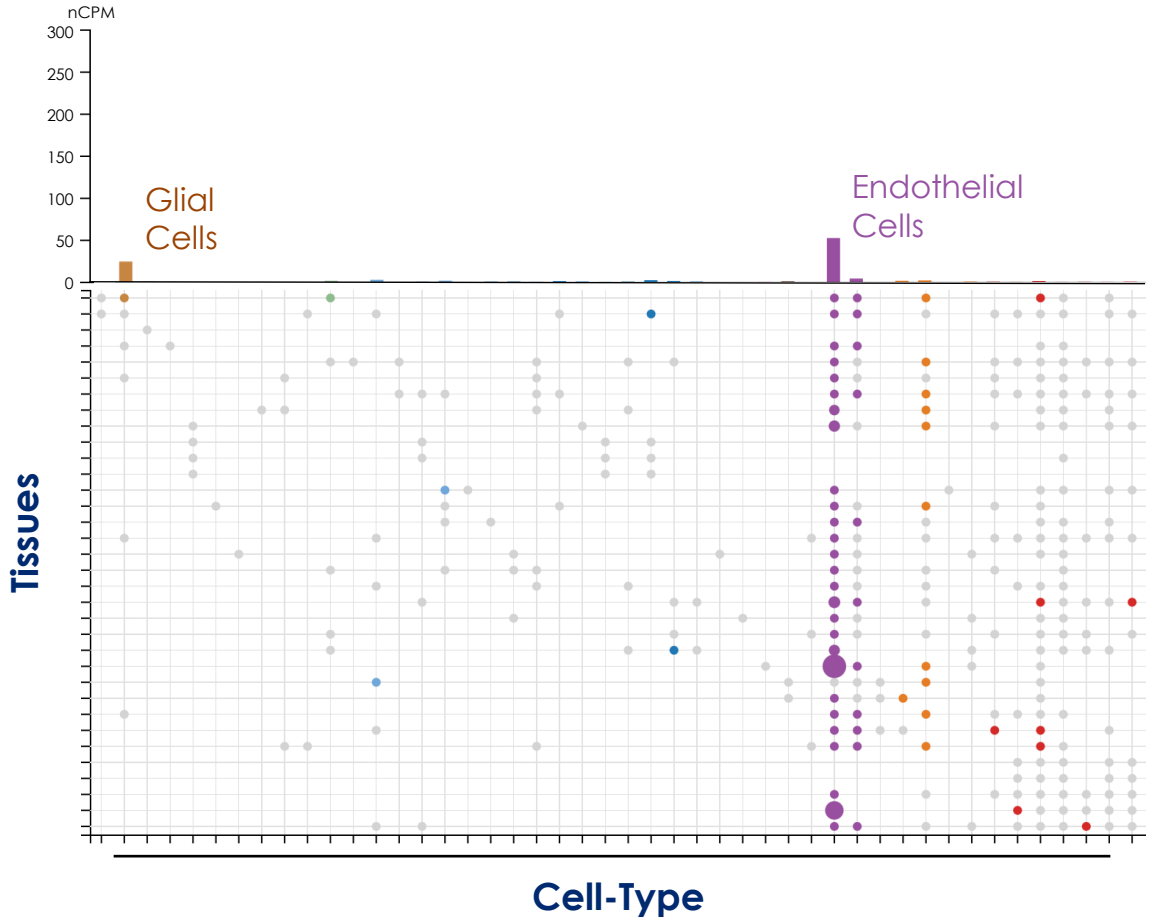
	VEGFR antagonism: Proven efficacy but poor long-term safety	**APJ antagonism:** Potential for durable efficacy without VEGFR toxicity
Selectivity	VEGFR and AKT signaling broadly required across adult tissues and vascular beds	APJ is endothelial cell enriched and pathway is most active in pathological sprouting angiogenesis
Normal biological function	Central to vascular homeostasis, renal microvascular integrity and repair biology	Low baseline activity in quiescent adult vasculature
Signaling pathways activated		

APJ Expression is Endothelial-Enriched While VEGFR2 Shows Broader Multi-Tissue Expression

VEGFR2 appears in more tissues leading to broader on-target biology



APJ is enriched in endothelial cells for more selective targeting



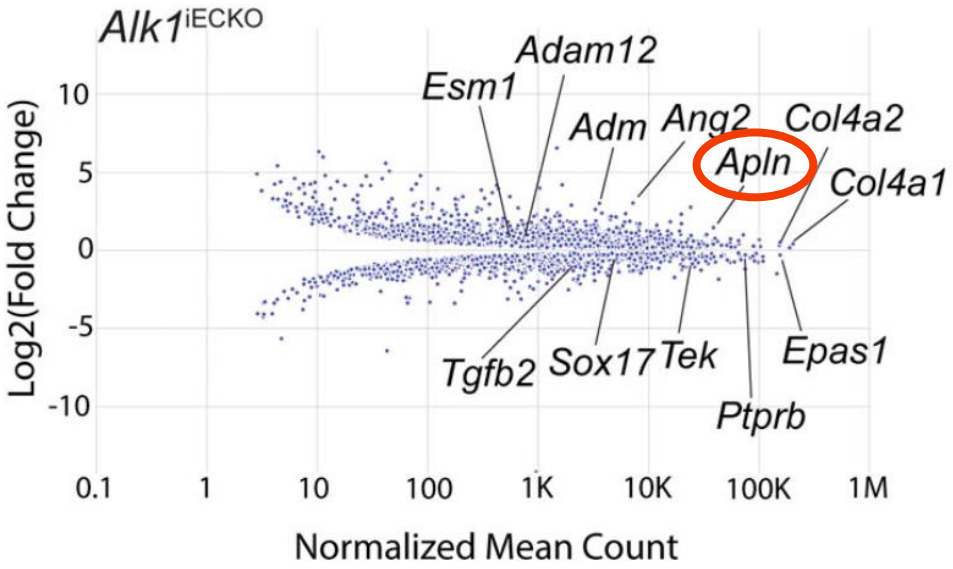
TECTONIC Therapeutic

Apelin is Upregulated in Endothelial Cells in Mouse Models of HHT

Potential for local apelin/APJ pathway activation in disease

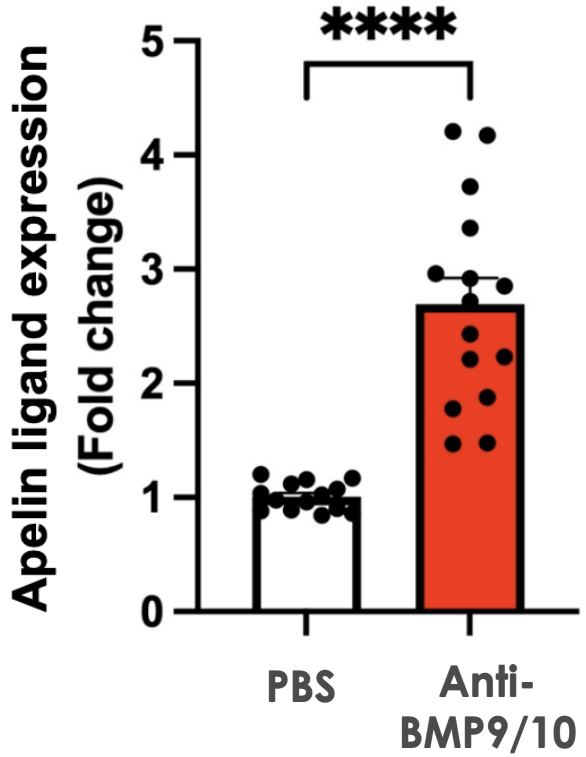
Shared angiogenic gene signature across HHT models

Apelin highly upregulated in endothelial cells in ALK1 KO mice[1]



EC transcriptomics: iALK1-KO vs f/f controls

Apelin expression upregulated in HHT BMP9/10ib model



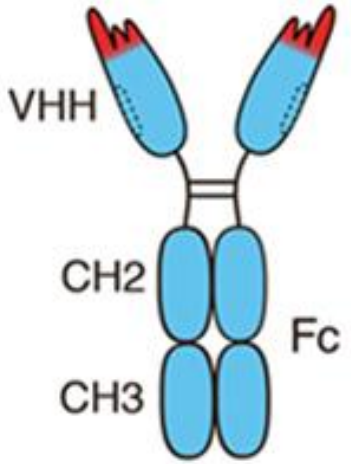
[1]Zhou et al ATVB 2023
****=p<0.0001 one-way Students t-test

TECTONIC Therapeutic

TX2100 is a Highly Potent and Selective Human APJ Antagonist

Low-nanomolar potency at human APJ with >1,000-fold selectivity vs. related GPCRs

TX2100

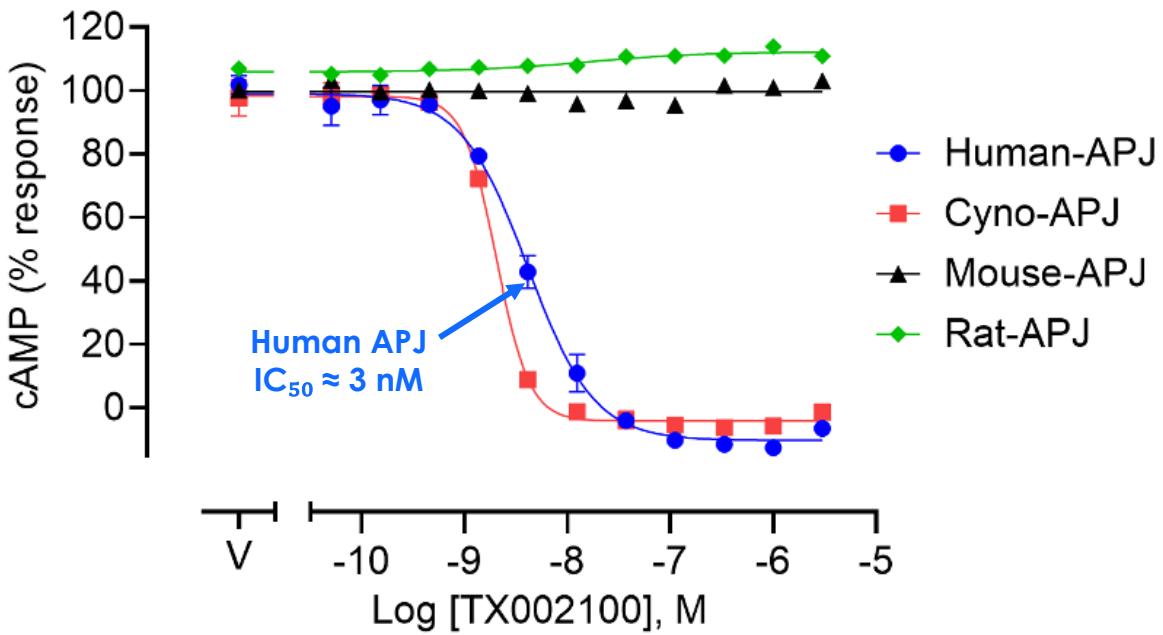


VHH-Fc fusion

- Highly specific, limits off-target toxicities
- Long half-life, less frequent dosing

Receptor	Pathway	IC_{50} (nM)
Human-APJ	cAMP	**3.1**
	β-arrestin	5.6
Most closely related GPCR	β-arrestin	**>1,000**
Mouse-APJ	cAMP	>1,000

TX2100 blocks cAMP signaling with nanomolar potency



cAMP measured *in vitro* in HEK293 cells

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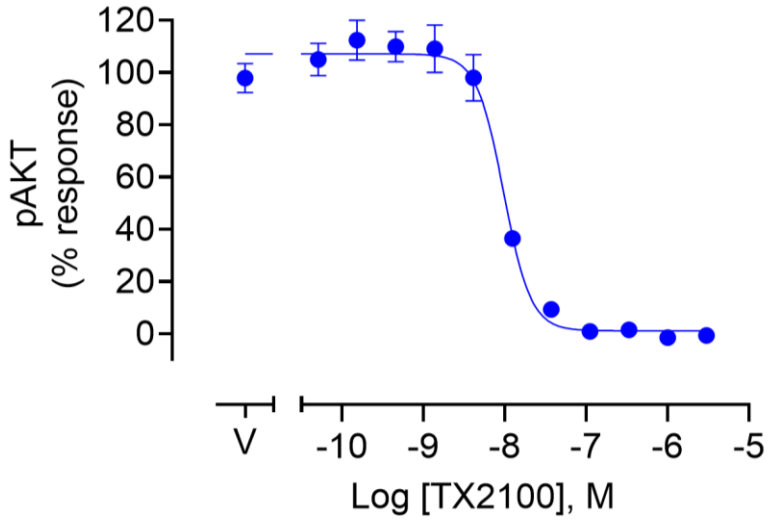
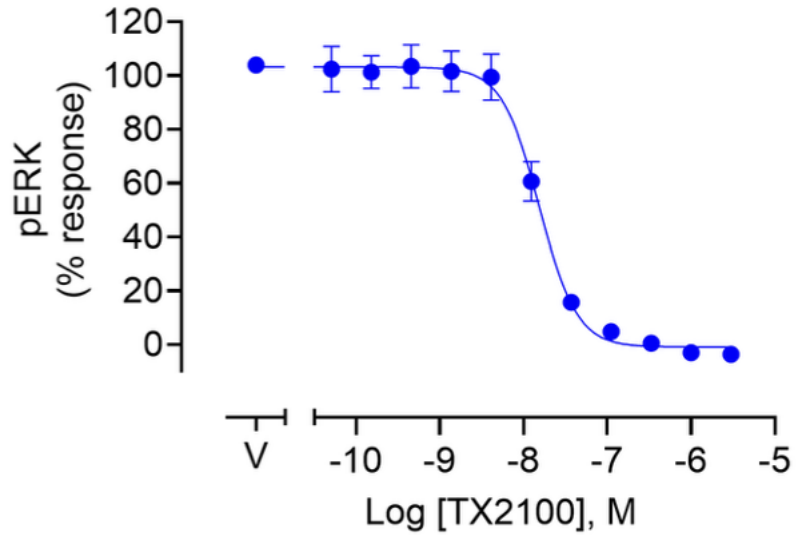
TX2100 Inhibits AKT and ERK Signaling Through APJ Antagonism, Blocking Pathways Important in Angiogenesis

AKT inhibition



ERK inhibition



APJ is primarily expressed in endothelial cells leading to AKT and ERK inhibition selectively in those cells

In contrast, VEGF/TKIs/AKT inhibitors are broadly expressed leading to systemic pathway inhibition which can result in safety and tolerability issues

AKT and ERK signaling measured *in vitro* in HEK293 cells

APJ Antagonism[1] Shows Robust and Durable Preclinical Activity in Two Complementary HHT Models

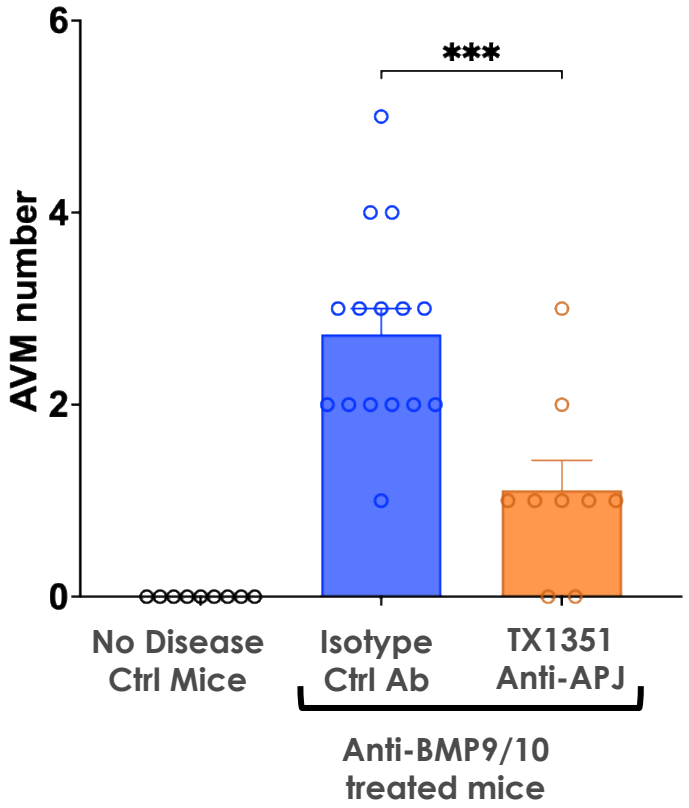
	Preclinical result of APJ antagonism
Neonatal anti-BMP9/10 Translational model of HHT generated by injection of anti-BMP9/10 antibodies into neonatal mice	• Reduced AVMs • Increased hemoglobin • Improved bleeding
Severe adult inducible ALK1-KO Most severe, clinically relevant model where disease is generated in a mature vascular system by tamoxifen-induced knockout of ALK1 in adult mice	• Durably increased hemoglobin (compared to anti-VEGF that waned over time) • Improved bleeding • Improved vascular architecture (reduced hypervascularization, abnormal dilation and AV shunts)

[1]TX1351 (surrogate anti-mAPJ VHH-Fc; potency matched to TX2100 against APJ) enables translatable *in vivo* testing

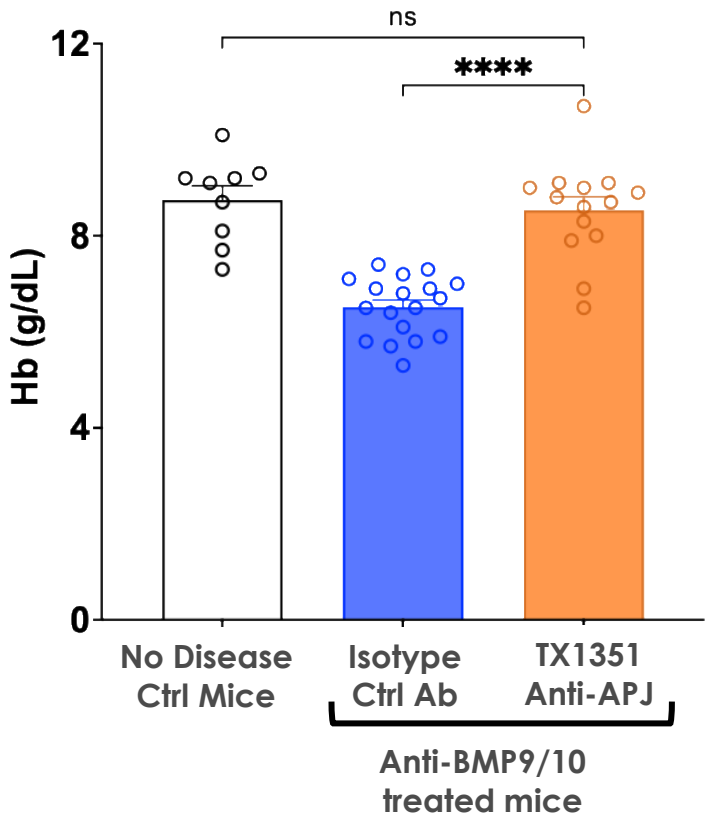
* AVMs = Arteriovenous Malformations

TX1351[1] Delivers Robust Disease-Modifying Phenotype in the Anti-BMP9/10 Model

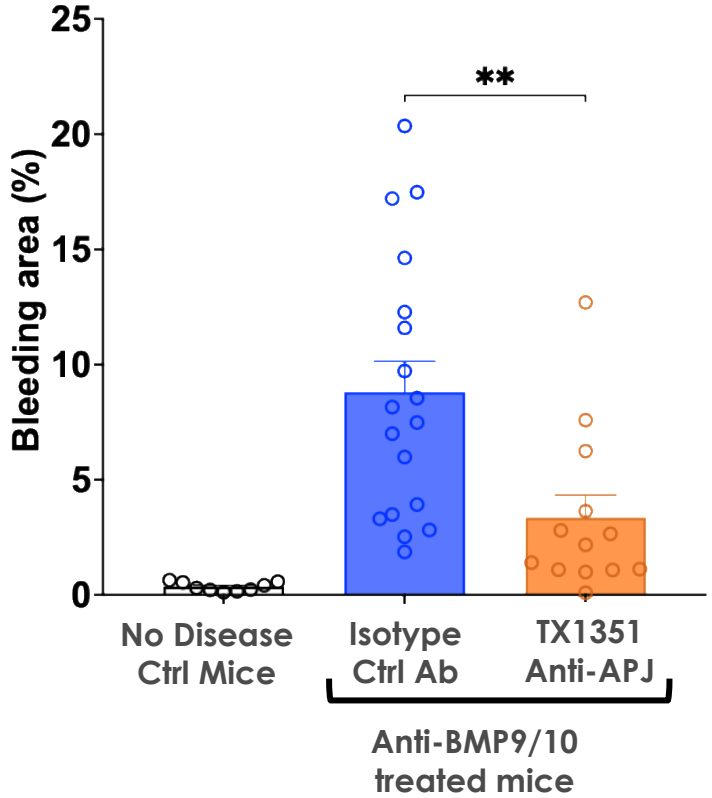
Decreases AVM formation



Increases hemoglobin



Reduces bleeding (retinal)



[1]TX1351 = TX2100 surrogate, anti-mouse VHH-Fc; Isotype Ctrl Ab = non-targeting VHH-Fc control
*=p<0.05, **=p<0.01, ***=p<0.001, ****=p<0.0001 One-way ANOVA followed by Tukey's multiple comparison test

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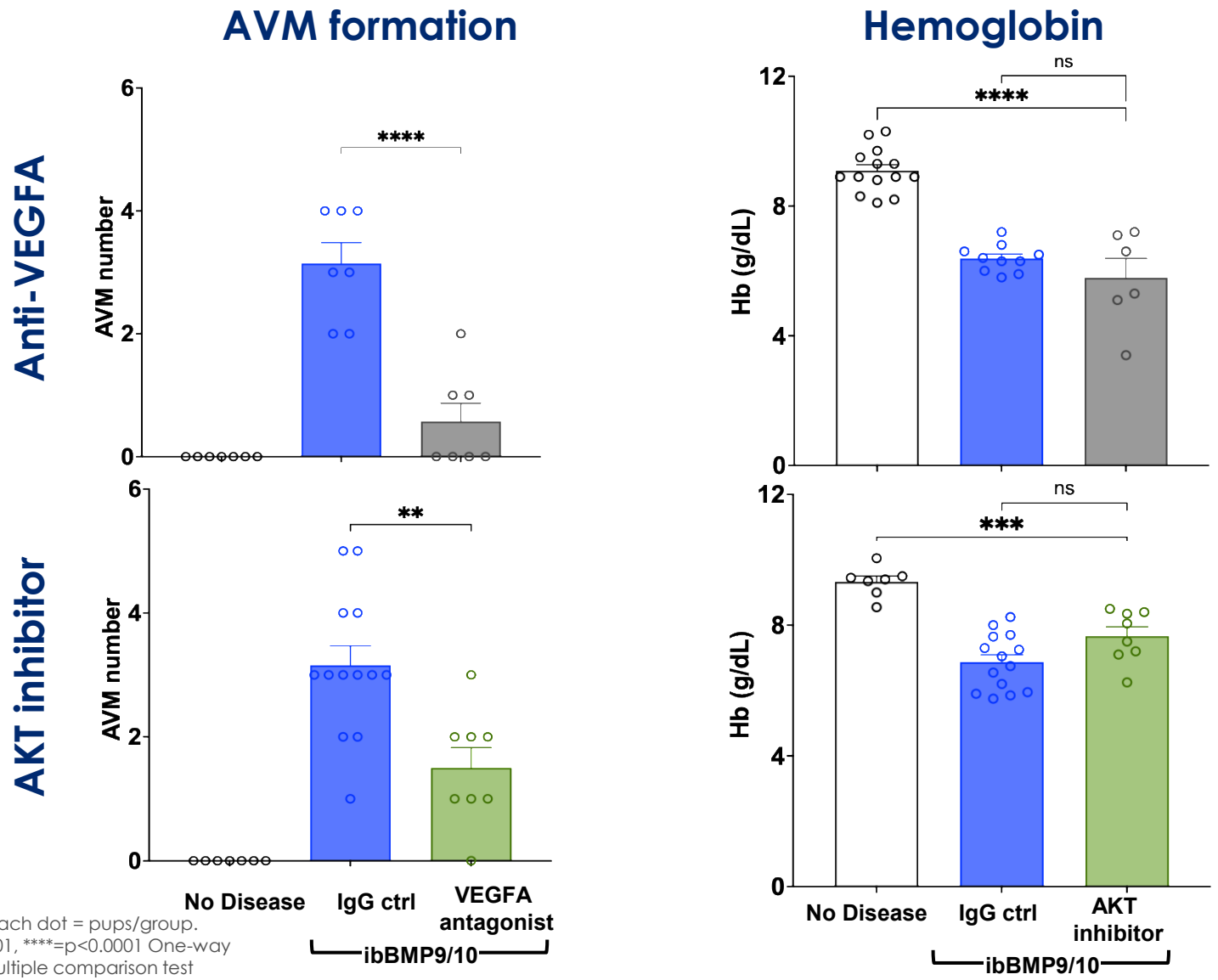
Anti-VEGFA, AKT Inhibition Provide Less Robust Disease Modification in the Anti-BMP9/10 Model



Data represent mean ± SEM; each dot = pups/group.
*=p<0.05, **=p<0.01, ***=p<0.001, ****=p<0.0001 One-way ANOVA followed by Tukey's multiple comparison test

Both mechanisms decrease AVM formation

Neither improved hemoglobin levels

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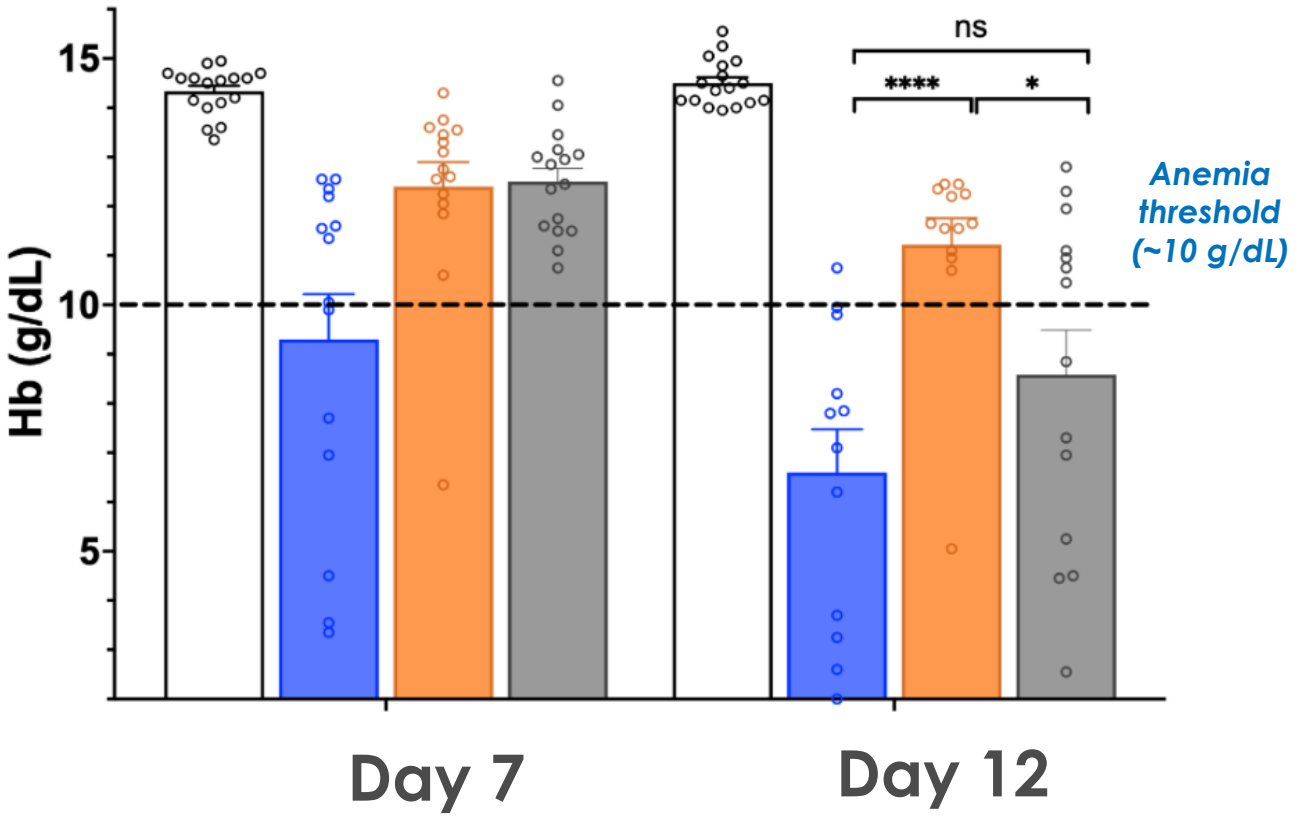
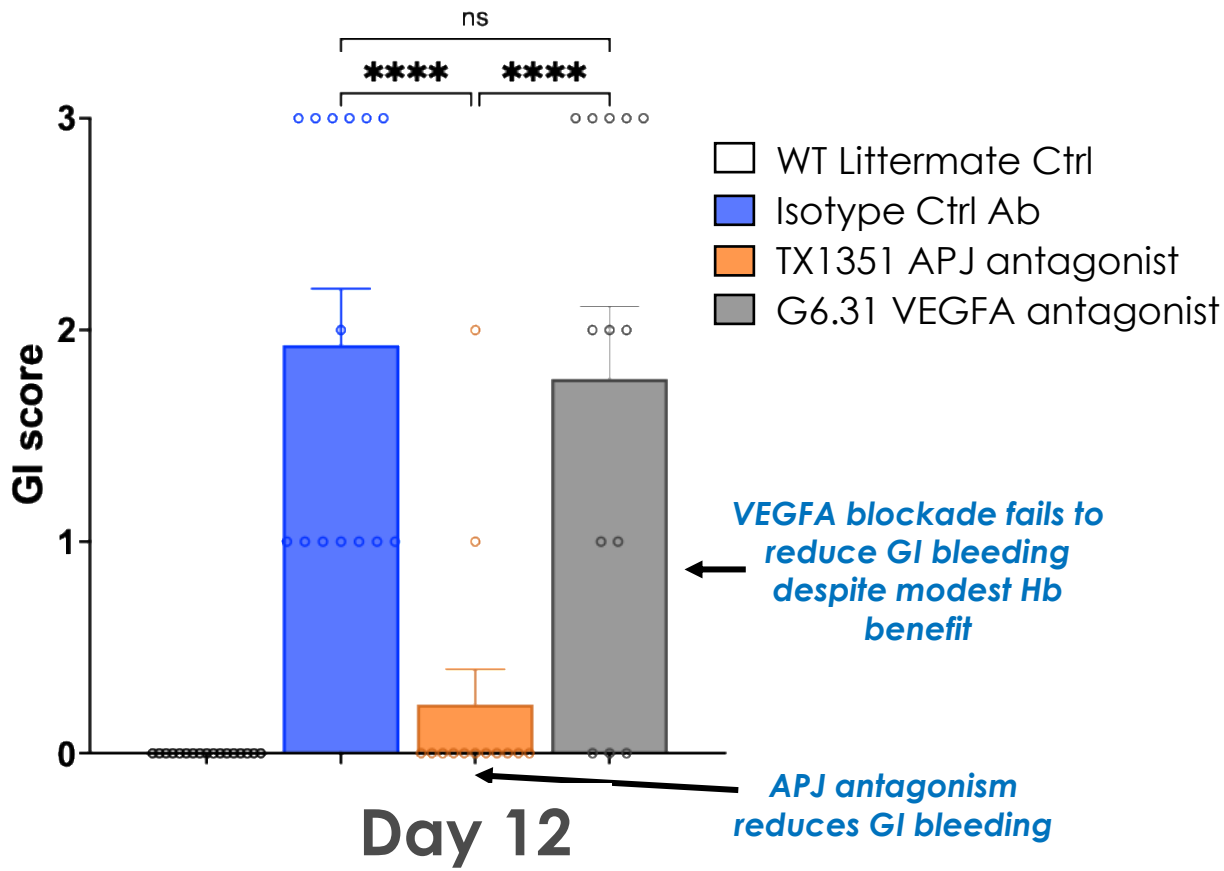
TX1351[1] Reduces Anemia & Bleeding in the iALK1-KO Model

APJ antagonism maintains durable benefits while VEGFA antagonism effects diminish over time

Hemoglobin Levels



GI Bleeding



[1]TX1351 = TX2100 surrogate, anti-mouse VHH-Fc; GI bleeding score measured on day 12
*=p<0.05, **=p<0.01, ***=p<0.001, ****=p<0.0001 One-way ANOVA followed by Tukey's multiple comparison test

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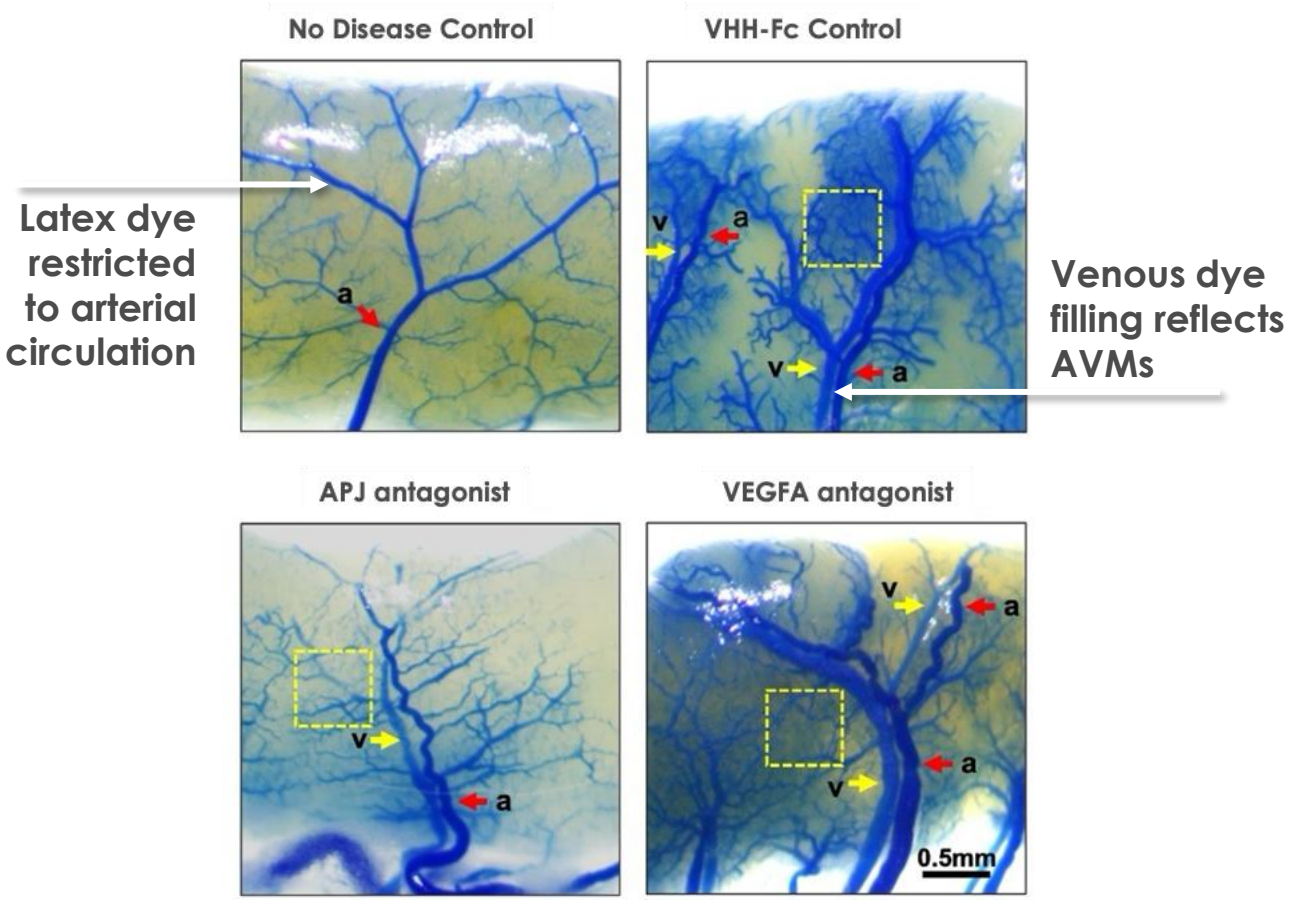
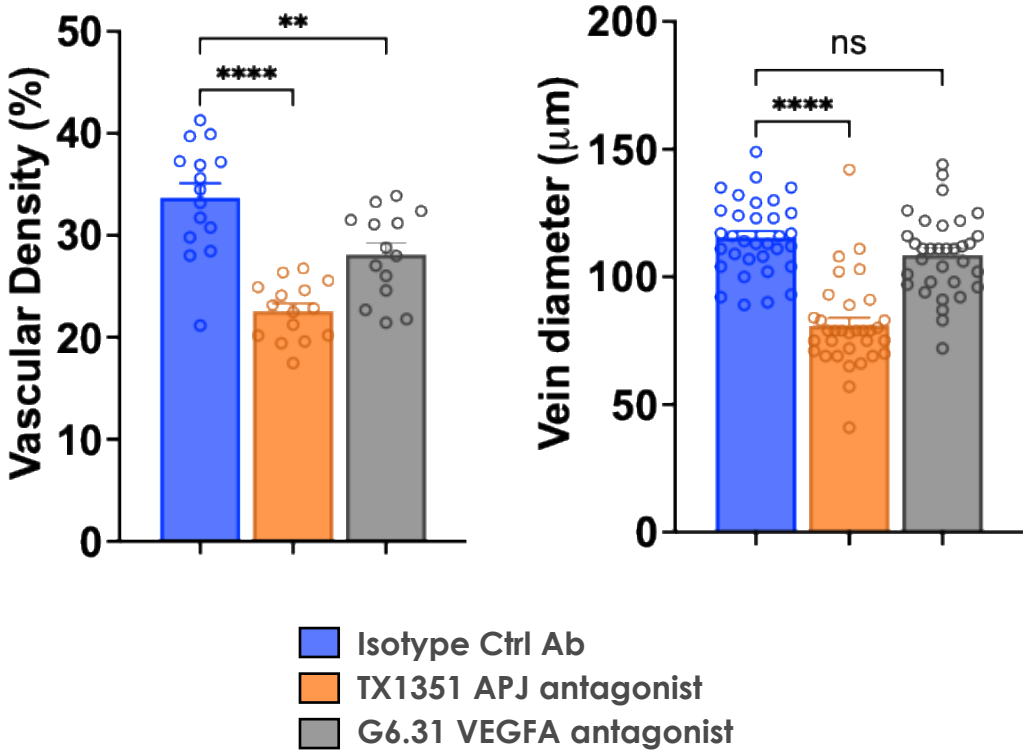
TX1351[1] Significantly Reduces GI Hypervascularization, Hemorrhage, and Vein Dilation in iALK1-KO Mice

APJ antagonism provides more complete vascular rescue than VEGFA antagonism



TX1351 restores vascular architecture toward normal in a severe HHT model



[1]TX1351 = TX2100 surrogate, anti-mouse VHH-Fc; Isotype Ctrl Ab = non-targeting VHH-Fc control
*=p<0.05, **=p<0.01, ***=p<0.001, ****=p<0.0001 One-way ANOVA followed by Tukey's multiple comparison test

Preclinical Program Did Not Show TX2100 or Target-Related Safety Signals

- **Apelin/APJ pathway has been studied mostly in the context of agonist pharmacology**
- **Clinical agonist programs did not show meaningful benefit**
  - Discontinued for lack of efficacy
  - Were generally safe and well tolerated, without major on-target liabilities

Previously reported physiological effects of apelin[1] and APJ antagonism were not reproduced in multiple in-house preclinical studies

- Blood pressure
- Renal function
- Platelets and bleeding time
- Glucose homeostasis
- Inflammation

Completed 13-week GLP toxicology study in non-human primates, showed no safety findings

- No CV, renal, muscle, or hematology findings
- No changes in glucose
- No BP or fluid balance issues
- NOAEL = 100 mg/kg/week (highest dose tested)

[1]Szokodi I Circ Res 2002, Coqueral D Am J Physiol 2021, Dray C Cell Metab 2008, Hus-Citharel A Endo 2014, Tatemoto K Reg Peptide 2001

TX2100 Preclinical Package Supports Phase 1a Clinical Development

- **Robust preclinical activity** across multiple, translatable models of HHT
- **Clean safety profile** in IND-enabling GLP toxicology studies, with no molecule specific or target-related safety signals
- **Patient-friendly SC formulation** identified
- **Drug product readiness** with favorable properties to support early clinical development

TX2100
Clinical Update

Marcella Ruddy, M.D.
Chief Medical Officer

Overview of TX2100 Clinical Development Plans

Ongoing Phase 1a first-in-human clinical trial in healthy volunteers

- Assess safety, tolerability and PK of single doses of TX2100
- Phase 1a first subject randomized in Feb 2026, expect topline results in Q4'26

Phase 1b clinical trial in patients with severe HHT

- Open label, multiple dose TX2100 study to assess safety and tolerability in patients
- Explore efficacy endpoints of epistaxis, anemia, and hematologic support

Phase 2 proof-of-concept clinical trial in moderate to severe HHT patients

- Randomized double blind placebo-controlled dose ranging study
- Assess safety and efficacy of TX2100
  - Improvement in epistaxis, anemia, hematologic support, and other HHT endpoints

Potential Opportunity to Expand TX2100 Patient Population

Anti-angiogenic mechanism of TX2100 offers opportunity to expand into other bleeding disorders caused by dysregulated angiogenesis

- Anti-angiogenic agents such as bevacizumab and thalidomide have demonstrated efficacy in treatment of other bleeding disorders caused by dysregulated angiogenesis

Preclinical data demonstrating activity of the APJ antagonist TX1351 in a non-HHT model of dysregulated angiogenesis-driven bleeding will be presented at a future scientific congress

TX2100: A Potential First-in-Class APJ Antagonist to Treat HHT

Validated approach

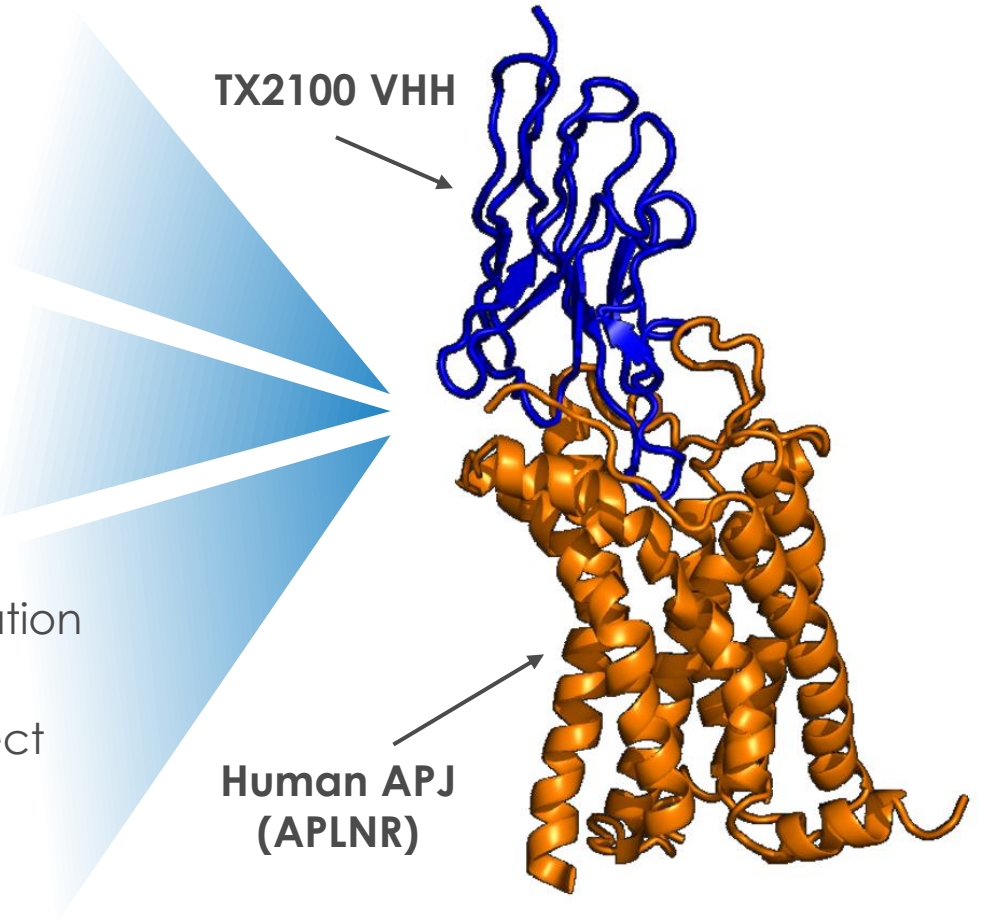
- Anti-angiogenesis improves bleeding/anemia in HHT
- Oncology agents can't be used chronically

Differentiated target / design

- APJ is endothelial-enriched + pathology-biased
- Built to capture anti-angiogenic benefit with improved safety

De-risked translation + path to value

- Preclinical activity in two validated HHT models + vascular normalization imaging
- Clean NHP GLP tox + durable PK → Phase 1a ongoing with first subject randomized in Feb 2026; Phase 1b and Phase 2 PoC planned



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Questions and Answers